Pediatric Supplement

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PEDIATRIC SUPPLEMENT

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BONE growth and development of CHILDREN*

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EVALUATION OF THE subject of development and growth of the skeleton in infants and children requires a rather broad survey of diseases inasmuch as so many of them disturb normal maturation of bone. Before these are discussed, however, it is necessary to understand some of the embryologic factors involved in bone formation and how these factors lead to the development of normal adult bone. We will explain some methods of determining whether or not normal maturation is occurring and will make special reference to the mensuration of long bones from radiographs. Then we will survey the diseases which have deleterious effects upon normal growing bone and what may be done to offset these effects.

Histogenesis of bone

The development of the skeleton is a most complex and a very long process. Because of these complexities and the length of time over which developmental changes occur, both congenital and environmental influences may have profound effects upon the developing skeletal system.

Bone begins to appear at about the eighth week of fetal life, and complete maturation of all bone structures is not attained until the individual is about 25 years of age. All bone develops from mesoderm, but not all directly from mesenchyme as do the membranous bones of the skull and face. The bony structures with which we are mostly concerned in this paper are those whose anlage is mesenchyme but which develop indirectly through an intermediary stage of cartilage. In this process there are: (1) endochondral bone formation in which spongy (cancellous) bone has arisen by deposition of lime in the matrix of the cartilage, disintegration and disappearance of cartilage cells, and appearance of osteoblasts and primordial marrow cavities and (2) periosteal bone formation in which compact bone develops peripherally about the cancellous bone by activity of the inner osteogenic layer of the perichondrium or periosteum. The compact bone is composed of the haversian systems: Buds of the osteogenic basal layer of the periosteum lay down secondary deposits of bone as concentric cylinders, the central axis of which is a tube containing blood vessels. Cells of the bone matrix line up about the buds which eventually separate from the periosteum. This represents, then, the beginning of a haversian system.

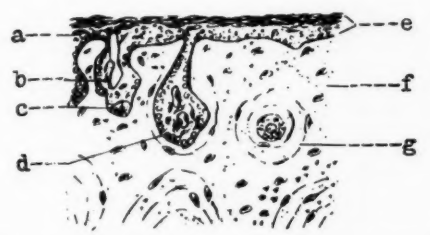


Fig. 1. Diagram of development of compact bone: (a) osteogenetic layer, (b) blood vessel, (c) osteoclasta, (d) osteoblasts, (e) periosteum, (f) bone matrix, (g) haversian system.

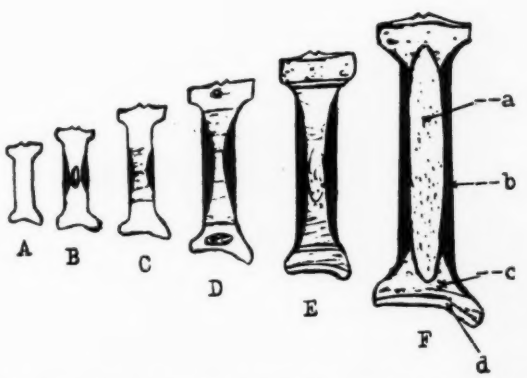


Fig. 2. Stages of long bone growth (A-F). Cross section of long bone; (a) marrow, (b) compact bone, (c) cancellous bone, (d) cartilage.

Growth of the long bones occurs in a rather distinctive way. Originally the bone is completely cartilaginous, but soon endochondral bone and compact bone invade this cartilage. Osteogenic tissue invades a portion of the cartilage remaining at the end of the long bone, at a later stage, forming the secondary center of ossification known as the epiphysis. This leaves a very

*From the Department of Radiology, Detroit Osteopathic Hospital, Detroit, Michigan. Presented at the twenty-fourth National Child's Health Conference and Clinic, Kansas City, Missouri, April 11, 1956.

active plate of cartilage between the epiphysis and the original bone shaft or diaphysis. The diaphyseal side of the plate is more active and produces cartilage which in turn ossifies and the bone lengthens. When adult length is attained this epiphyseal plate ossifies and the process ceases.

Skeletal maturation

The rate of appearance of the epiphyses and the time of their fusion with the diaphyses may be utilized in the appraisal of skeletal growth.

Charts are available which indicate the age at which the various epiphyses normally appear and when they fuse with their respective diaphyses. Such skeletal charts are quite useful to the radiologist interpreting radiographs of infants and children.

In assessing skeletal maturation several points must be kept in mind which may influence conclusions: (1) Skeletal maturation occurs more rapidly in the female than in the male so that at puberty there is 2 years' difference in terms of maturation. (2) Each child has its own individual growth rate. (3) Skeletal development is more variable from one individual to another near the onset of puberty than at other periods. (4) Skeletal development varies also with the type of population to which the individual belongs.

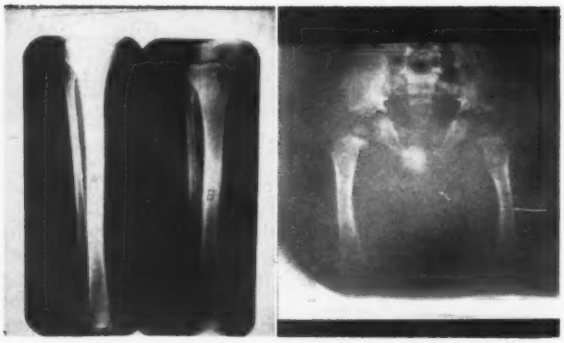
Various methods have been formulated for determination of bone maturity. Most of these deal with the counting of epiphyseal ossification centers from radiographs of all extremities, of the foot and ankle, or of the hand and wrist. The last method is rather widely used. It utilizes a radiograph of the wrist and hand and the counting the epiphyseal centers of the distal radius and ulna, the carpals, and all the epiphyses of the metacarpals and phalanges. There are thirty of these. Normally none of these centers are ossified at birth. At 3 months of age, one or two are ossified. At 1 year, three to four are ossified; at 2 years, twelve to fifteen; and at 3 years, nineteen to twenty-two in the male, and twenty-three to twenty-four in the female. Tables showing the age of appearance of these ossification centers are available in most pediatric and roentgenologic texts. Using the above figures as an average for normal bony development, one can judge whether skeletal maturation is slow or accelerated.

Radiographic technic

Scanography is a useful technic for accurate measurement of the length of long bones directly from a radiograph. Measuring length directly from conventional radiographs is unreliable because of distortion. Scanography is a method of making films by the use of a narrow slit beneath the x-ray tube in such a manner that only a narrow line or sheet of x-rays is used while the x-ray tube moves over the object. Thus all the rays of the central beam pass through all areas of the part at the same angle. Figure 3 shows two exposures of tibia and fibula. Figure 3a is a conventional radiograph showing distortion and elongation of the bones as compared to the scanographic exposure, Figure 3b, which is without distortion; actual bone length may be measured directly from the film.

Another procedure which is readily usable without special equipment is that of marking the skin directly over the epiphyseal plate at either end of a long bone and making a coned exposure directly over each area on only one film while the film and the patient remain in exactly the same position. When a bone is too long to be filmed on conventional-size film, this same procedure may be used with a separate film for each exposure and a long perforated rule in the same position beside the extremity during each exposure.

Fig. 3. Scanography: (A) conventional radiograph, (B) scanograph of same part. Fig. 4. Achondroplasia.



Developmental malformations

Achondroplasia.—This condition is a developmental disorder of the extremities due probably to arrested endochondral bone formation and leading to a form of dwarfism. It is suspected that this is an inherited trait or simply a mutation. It is characterized by a trunk of fairly normal length, but the extremities are short and the legs are usually bowed. Roentgen studies show all of the diaphyses of the long and short tubular bones to be quite short, and there is considerable flaring of the metaphyses. Development of the periosteal (compact) bone is normal, but a disturbance of endochondral ossification results in deficient longitudinal growth of bone. There are other skeletal changes such as lumbar lordosis, prominent mandible, and a heavy bony structure about the sella turcica.

Congenital Osteochondrodystrophy.—This is a rather rare type of familial dwarfism involving, for the most part, the epiphyses. Marked kyphosis is usually present about the dorsolumbar juncture with wedge-shaped vertebrae. There are rib and chest deformities. In the extremities the capital femoral epiphyses are fragmented or absent. Epiphyses are crenated, and there may be calcifications in cartilaginous structures. The changes in the capital femoral epiphyses may resemble those of Legg-Calvé-Perthes' disease, but the spinal changes will establish the diagnosis.

Osteogenesis Imperfecta.—This is a disease of unknown cause and is characterized by abnormal fragility of bones. The severity of the disease varies greatly, from fractures occurring in utero (osteogenesis imperfecta congenita) which may result in stillbirth, to fractures occurring several years after birth (osteogenesis imperfecta tarda). In the severe forms the long bones, ribs, and clavicles are broad and short, showing many areas of callous formation as evidence of in utero fractures. Later, the long bones are very slender while epiphyses appear large in comparison with the diaphyses, and may become bowed. The calvaria is very thin and pliable. In milder cases, there is muscular weakness, often blue sclera, and some degree of dwarfism. Deafness is common. In the mildest forms, the fragility may be outgrown. This disease is apparently a deficiency of the subperiosteal osteoblasts and thus of periosteal compact bone which forms the cortex of the diaphyses.

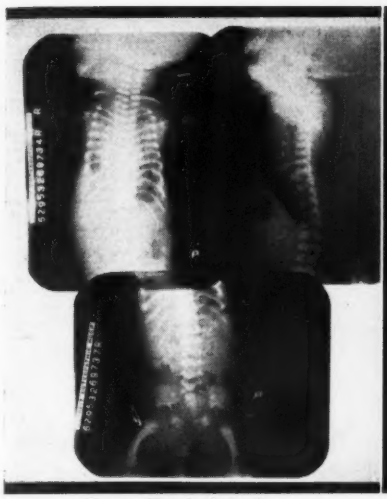


Fig. 5. Chondrodystrophy.

Fig. 6. Osteogenesis imperfecta.

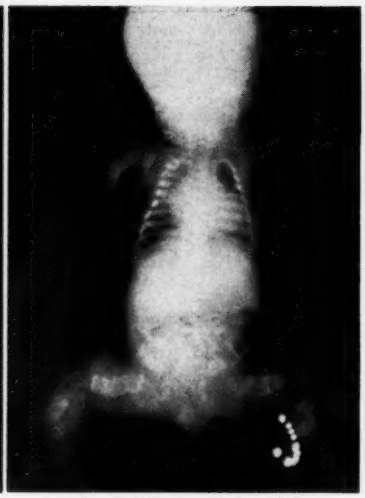


Fig. 7. Osteo genesis imperfecta.

Metabolic disorders

Hypervitaminosis A.—Excessive administration of the fat-soluble vitamin A is known to produce toxic effects. It is mainly a disturbance of infants who have been given large amounts of various vitamin products in concentrated forms by parents who have become overconscious of vitamins through reading enthusiastic advertisements. When this takes place the fasting serum vitamin A levels increase tremendously. Marked periosteal proliferations occur, usually in the mid-diaphyseal portions of the long bones, producing tender bony prominences over the extremities, ribs, and clavicles. Deossification is usually noted in the phalanges, and complete or incomplete pathologic fractures may occur. Infantile cortical hyperostosis is a similar disease of unknown origin in which serum vitamin A level is normal.

Rickets.—

This deficiency disease is an anabolic disorder of the inorganic constituents of bone due to deprivation of ultraviolet radiations, deficiency of vitamin D, and constitutional susceptibilities to the disease. The ingested vitamin D may not be absorbed from the gastrointestinal tract, as in celiac diseases with steatorrhea, in hepatitis, or in congenital stenosis of the bile ducts in which normal absorption of fats, which is necessary for the absorption of vitamin D, does not occur. In certain cases, that is, in renal rickets, the vitamin is absorbed but is not normally utilized, because of renal dysfunction.

Radiographically all bones may show involvement, but especially those areas of active growth of bone. Widespread deossification, thinning of the cortices of bone, and flaring and tufting of the metaphyses can be demonstrated. There is often bowing of the long bones, and fractures may occur. In the healing stage there is considerable periosteal overlay with reossification, particularly along the concave side of a long bone.

In the treatment of some refractory cases of rickets, considerable amounts of vitamin D must be given the child. Some cases of refractory rickets have been incorrectly diagnosed as osteogenesis imperfecta when normal dosages of vitamin D do not produce any response. Caution is required to avoid inducing hypervitaminosis D and its toxic effects.

Hypervitaminosis D.—The toxic effects seen in this state are due to overadministration of vitamin D,

†Material for Figures 8 through 12 and 16 through 18 was supplied through the courtesy of Dr. Paul T. Lloyd of Philadelphia.

Fig. 8. Rickets, active in ribs.†

Fig. 9. Rickets, under treatment.

Fig. 10. Rickets healing.

Fig 11. Scurvy.

Fig. 12. Lead poisoning.

another fat soluble vitamin. These effects include deposition of calcium in soft tissues of tendon sheaths, joint capsules, arteries, parenchyma of the kidneys, and costal cartilages. Pathologic fractures may occur.

Scurvy.—This disease is associated with a deficiency of vitamin C, and the pathologic changes occur primarily because the increase in permeability of the vascular walls results in hemorrhages or escape of serum and various blood constituents. Secondarily, there is hypervascularization resulting in generalized deossification and thinning of the cortices of the long bones. There are condensations in the metaphyses and about the periphery of the epiphyses, and there may also be ballooning out of the periosteum due to subperiosteal hemorrhage.

Heavy metal depositions

As a result of ingestion of substances containing lead, bismuth, phosphorus, and fluorine, these heavy metals may combine with normal bone calcium and phosphorus, or they may produce bone deficiencies by "salting out" substances in the gastrointestinal tract before they can be normally absorbed. Rickets may result. Radiographically, the long bones manifest some of the following: ribbonlike condensations in the metaphyses of the long bones or transverse striations of condensations higher up along the diaphyseal shaft. Occasionally there is an increase in density of fecal matter in the bowel when lead or bismuth substances have been ingested. Lead and bismuth poisonings occur more often in infants whereas phosphorous and fluorine poisonings, which progress more slowly, usually occur in older children.

Endocrinopathies

Hypothyroidism.—With decreased or absent thyroid secretion there is a reduced rate of cellular metabolism. With respect to growing bone, the effects occur for the most part in the extremities, resulting in a delay in the appearance of the secondary centers of ossification. The diaphyses are relatively short and stubby. When the secondary centers of ossification do appear they are often segmented or stippled. There may be condensations in the metaphyses which may confuse the differentiation of this disease. Response to thyroid treatment is rapid.

Hyperthyroidism.—With excessive thyroid secretion in children the reverse of the above is noted. More rapid development of the secondary centers of ossification and early closure or ossification of the epiphyses occur. This may be secondary to overstimulation of the gonads, however. These early closures of the epiphyses result in reduced long bone lengths.

Hypoparathyroidism.—In this disorder there is early closure of the epiphyses, resulting in short stature, stubby hands and feet, and hypoplastic dental development.

Hyperparathyroidism.—All portions of the long bones are demineralized. Hyperparathyroidism is a slowly progressive disease, and in older children cystic rarefactions, pathologic fractures, bowing and angulation deformities, and kyphosis and scoliosis are observed radiographically. Scout films of the abdomen quite often show urinary stones.

Pituitarism.—Adenomas of the chromophobe type give rise to pituitary dwarfism. Adenomas of the acidophilic type give rise to giantism.

Gonadal Dysfunction.—Changes due to gonadal dysfunction are usually seen in the first decade of life. Hypersecretion of the gonads leads to early appearance of the secondary centers of ossification and usually to premature closure of the epiphyses. Hyposecretion of the gonads leads to late appearance of the secondary centers of ossification and delayed closure of the epiphyses. This type has been related to certain forms of nonspecific osteochondritis, although there may be varied inter-relationships with the function of other endocrine glands.

Bone infections

Osteomyelitis.—

Osteomyelitis is usually caused either by a hematogenous bone infection from a primary focus of infection elsewhere in the body or by infection introduced locally from compound fractures or regional soft tissue cellulitis. The sites of predilection are in most cases

Fig. 13. Pituitary tumor.

Fig. 14. Osteomyelitis.

Fig. 15. Tuberculosis of kneejoint.

Fig. 16. Congenital syphilis—gumma of bone.

Fig. 17. 2 weeks later than in Fig. 16.

Fig. 18. Congenital syphilis of tibia.

Fig. 19. Osteogenic sarcoma.

Fig. 20. Poliomyelitis.

in the extremities, almost always the lower. The course of the disease may be divided into four stages: acute, subacute, chronic, and residual.

The infective process usually begins in the epiphyseal side of the metaphyses and progresses to the cancellous part of the bone. After the cancellous bone has been permeated, the periosteum is penetrated and abscess formation occurs in the subperiosteal space. There is evidence of a lytic bone destruction process early, followed later by osteosclerosis and periosteal proliferation. Sequestration occurs, and a portion of avascular and sclerotic cortical bone is demarcated from the lytic process. New bone, involucrum, is formed just outside the sclerotic portions of the original cortical shaft. The process does not pass beyond the epiphyseal boundary unless the epiphysis is closed or unless the involved metaphysis lies within the confines of the joint capsule.

Osteomyelitis of the hip joint is a classic example, and in this joint pyarthrosis develops with secondary involvement and luxation of the epiphysis and destruction of the epiphyseal cartilage. In this case there will be shortening of the long bones. However, elongation of the long bone may occur as well if the metaphyseal involvement is not intracapsular and the epiphyseal cartilage is not destroyed. This is due to increased growth at the undisturbed epiphyseal cartilage from the hyperemia accompanying the infection.

Tuberculosis.—With respect to bone growth, tuberculosis may produce elongations and shortenings of the involved long bones as does osteomyelitis and by the same mechanisms.

Infantile and Juvenile Syphilis.—In syphilis of these types, the epiphyses and epiphyseal cartilages are usually not involved except in rare cases of the juvenile type in which Charcot's joints develop and thus growth in length of the long bone is reduced. The most common and characteristic findings are diffuse or localized subperiosteal cortical thickening, transverse condensations in the metaphyses, and infractions or erosions of the inner aspects of the metaphyses of the femur and tibia.

Neoplasms

Osteogenic Sarcoma.—This is a relatively rare malignant neoplasm originating from the inner layer of the periosteum and the endosteum and appearing in children in the juvenile age group. Eighty per cent of these lesions occur in the lower extremity about the knee in the metaphysis. There may be osteogenic and osteolytic changes. The former will likely consist of cortical thickening, periosteal elevation, perpendicular striations, and breakthrough and invasion of the regional soft tissues. The osteolytic type presents areas of radiolucency.

Ewing's Tumor.—This is a rapidly growing malignant lesion which most commonly involves the diaphyses of the long bones, producing condensations of bone, periosteal layering (onion skin appearance), and invasion of the soft tissues, and occasionally resembling osteogenic sarcoma.

Leukemic Infiltration.—Bone replacement by blood-forming elements may occur in lymphatic or myelogenous leukemia in children from 1 to 10 years of age. The changes are seen in the long bones and are characterized by thinning of the cortices, metaphyseal deossification, coarse trabecular structures, possibly periosteal overlay, and pathologic fractures.

Neuroblastoma.—Metastatic extension of a neuroblastoma may appear radiographically to be very similar to leukemic infiltrations and blood dyscrasias.

Blood dyscrasias

Cooley's Anemia.—Thalassemia is a rapidly progressing anemia in youngsters usually under 6 years of age. There are bone changes in the skull and long bones. In the latter there are thinning and expansion of the cortices and coarsening of the trabeculae similar to the changes seen in leukemic infiltrations.

Sickle Cell Anemia.—The findings in sickle cell anemia are much the same as in leukemic infiltrations and in Cooley's anemia. Final differentiation is often dependent upon blood counts and morphology.

Arthropathies

Rheumatoid Arthritis.—Rheumatoid arthritis has been reported in children as early as 3 years of age. One of many theories as to its cause is that it is a chronic allergic type of arthritis. Radiographic changes include fusiform periarticular swellings, deossification of the epiphyses and metaphyses, narrowing of the joint spaces, subluxations, and ankylosis late in the disease.

Hemophilia.—This disorder is a hemorrhagic diathesis, the bleeding affecting the weight-bearing joints of the lower extremities. When hemorrhage occurs beneath or near the articular cartilages, the result is destruction of the cartilage with osteoarthritic

Fig. 21. Slipped epiphysis.

Fig. 22. Slipped epiphysis, internal fixation.

Fig. 23. Growth stimulation.

Fig. 24. Growth suppression.

changes. Expansion of the joint capsule may be seen early, whereas regional deossification, irregularities of the articular surfaces, and cystic erosions of the articular cortices are seen later.

Poliomyelitis.—Deficiencies of motor nerve function, as in poliomyelitis, result in muscular weakness and imbalance. This leads to deossification and to degeneration of articular cartilages. Articular surfaces become irregular and flat. Subluxations and dislocations may result. Usually also, as a result of the above changes, there is underdevelopment of the long bones involved.

Paraplegic Joints.—Certain spinal neurologic lesions produce profound disruption of normal articular contours with deossification, periosteal proliferation, and osteoarthritic changes with fragmentation of the articular surface cortices.

Traumatic lesions

Fractures.—Of interest to this paper are those fractures and dislocations which involve the epiphyses of the long bones by damaging the epiphyseal cartilage to the extent that there is premature closure of that epiphysis. In this event the full growth in length of the involved bone is not attained. Also in fractures of the femur or tibia, for example, overlapping of fragments may well result in relative shortening of the involved bone as compared to the opposite side. If this overlap is not too great, however, the epiphyses at either end of the bone may become active enough to compensate for this shortening.

Legg-Calvé-Perthes' Disease.—This disease, aseptic necrosis of the capital femoral epiphysis, is believed to be related to various endocrine disorders, but the metabolic disturbances which result in rickets, and traumatic or strain factors may also be etiologic factors. Regardless of the cause of the disease, aseptic necrosis results from reduced vascularity to the epiphysis. Inactivity of the part for months or years will allow the epiphysis to undergo some degree of regeneration. Early changes include bone condensations within the epiphysis followed by fragmentation.

Slipped Capital Epiphysis.—Gonadal endocrinopathy and strain upon the cartilage of the femoral neck due to weight bearing by hypersthenic children are generally believed to be etiologic factors. Aseptic necrotic changes occur in the femoral neck with disturbance of the epiphyseal cartilage.

Control of long bone growth by surgical means

Other than specific therapy, if any, for the particular disease process, treatment is surgical, designed either to stimulate or to suppress long bone growth in length. If, for example, there is retarded growth of an extremity, as in poliomyelitis, metal screws or ivory pins can be inserted into the metaphysis parallel to the epiphyseal cartilages of the distal femur and proximal tibia. The presence of these foreign bodies gives rise to hyperemia and greater growth activity at the epiphysis. Conversely, if there has been greater growth in length of a long bone, which may occur in certain forms of osteomyelitis, staples may be used to bridge the epiphyseal cartilage and anchor the epiphysis to the metaphysis. This will halt growth in length at that point. Then if serial scanographic studies are made throughout the months and years of development following, the progress of treatment can be accurately determined.

Summary

We have attempted to survey the many factors which are involved in the normal development and maturation of bone and the many congenital and acquired disease processes which may alter this growth. Also we have outlined various procedures by which the radiologist may aid the clinician in assaying the stage of skeletal growth in children, and we have mentioned methods by which long bone growth may be controlled by surgical intervention.

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The ROLE of the ADRENALS during STRESS in infancy and childhood*

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HIDDEN UNDER THE diaphragm in the fat surrounding the upper pole of each kidney is a body of tissue whose small size belies its great importance. When consideration is given the fact that this small gland is the source of substances which are essential to the maintenance of life, it can be realized how appallingly inadequate and surprisingly recent is most of our knowledge of the adrenal and its functions and effects.

Investigation of the adrenals as the possible source of vital secretions began in 1855 when Addison noticed a connection between certain anatomic abnormalities of the gland and the clinical syndrome which now bears his name. Relatively little was gained by these investigations, however, for attention was focused on the medulla and its one secretion known at that time, epinephrine.[1]

Study of the adrenals was given new impetus in 1927 when a cortical hormone was discovered which gave promise of being the key which would unlock the secret of the adrenals. During the past 25 years, numerous experiments and investigations have been made, and knowledge of the adrenals, and especially of the adrenal cortex, has grown tremendously. Yet in spite of much study and research, there are still a great many questions that remain essentially unanswered.

One of these questions regards the role of the adrenal during stress in infancy and childhood, and an attempt has been made in the preparation of this paper to sift through a vast amount of experimental data and other literature in the hope of finding at least a partial answer.

In order to understand better the changes taking place in the adrenals at various periods of life, it may be well to review what is known of the origin and histology of these glands and to evaluate their role in infancy and childhood by a comparison with what has been learned of the well-developed adult organ.

At about the sixth week of fetal life, cells of the splanchnic mesoderm begin to proliferate and form a mass which is destined to become the adrenal cortex. Chromaffin cells, arising from the neural crest, invade this mass to form a central core or medulla.[2] Growth is rapid during fetal life, and at birth the adrenal is relatively very large, being about one-third the size of the kidney.[3]

The adrenal of the newborn consists of the centrally located medulla surrounded by a wide layer of well-developed cell cords, the temporary or fetal cortex, or X-zone, which in turn is encapsulated by a thin layer of more undifferentiated cells, the true permanent or adult cortex. Immediately after birth, or possibly in late fetal life, the temporary zone begins to degenerate and rapid involution takes place.[2] It is possible that only the parenchymatous cells degenerate and rather quickly disappear, leaving only connective tissue, which forms a stroma for the more slowly developing cells of the zona reticularis. This involution is normally completed by about 1 year of age or earlier.[4,5]

The outer true zone of the infant adrenal resembles the adult zona glomerulosa except that it is well developed, whereas the latter is a relatively narrow layer consisting of groups of small cells, somewhat similar to kidney glomeruli.[5] The zona fasciculata begins to make its appearance in about the second postnatal week, and the zona reticularis can be definitely recognized at about the third month.[4]

In the normal adult gland the various zones are clearly discernible, and some of their functions have been determined. The large cells of the cortex, filled with a lipid substance and called foam cells, form irregular strands surrounding the sinusoids. These cells secrete their hormones directly into the sinusoids which are filled with blood from the adrenal, renal, and splanchnic arteries, and empty into the adrenal vein.[6]

Extracts from the adrenal cortex have been found to contain some thirty different crystalline compounds which have been isolated and identified chemically. Of these, five have been shown to have physiologic activity and are known to occur in the normal human adrenal. These are compounds A (11-dehydrocorticosterone), B (corticosterone), E (17-hydroxy-11-dehydrocorticosterone), and F (17-hydroxycorticosterone or hydrocortisone), and aldosterone (electrocortin). An amorphous fraction remaining after all crystalline material has been removed from adrenal extracts has also been found to have great activity in the prevention and treatment of adrenal insufficiency.[5]

Following stimulation of the human adrenal cortex by corticotropin, compound F is found in large quantities in the plasma. It is also the major free circulating adrenocortical hormone even before such stimulation, and over 90 per cent of the activity in the crystalline fraction has been ascribed to this steroid. It is thought that the breakdown of hydrocortisone may

*This paper received the Pediatric Award (1956) offered by the staff of the Pediatrics Department, College of Osteopathic Physicians and Surgeons, Los Angeles, California, for the outstanding paper written by a member of the Senior Class of the College. Dr. Byberg is now interning at the Los Angeles County Hospital.

give rise to other hormones, such as cortisone and corticosterone.[6] Compound B and aldosterone are found in relatively small quantities.[7]

According to Harper,[8] all of the corticosteroids mentioned above

> . . . increase the absorption of sodium and chloride by the renal tubules and decrease their excretion by the sweat glands, salivary glands, and the gastro-intestinal tract. However, there is a very considerable difference in the extent of this action by the various corticoids. Hydrocortisone (compound F) shows the least sodium-retaining action, whereas aldosterone is extremely potent, being at least 1,000 times as effective as hydrocortisone in electrolyte regulation. Accompanying the retention of sodium, there is increased excretion of potassium, caused mainly by exchange of intracellular potassium with extracellular sodium. . . . In maintaining the life of the adrenalectomized animal, aldosterone is more potent than any other known steroid.

The adrenal cortical hormones have been classified under three main types: (1) the water and electrolyte (or desoxycorticosterone acetate-like) hormones, (2) the glycogenic (S, sugar, or cortisone-like) hormones, and (3) the androgenic (N or protein anabolic) hormones. Aldosterone and desoxycorticosterone (usually used as the synthetic acetate) are the chief salt- and water-regulating corticoids. Examples of the glycogenic or metabolic corticosteroids are compounds A, B, E, and F, all of which are 11-oxysteroids.

Adrenosterone, with about one-fifth of the androgenic activity of androsterone, has been isolated from beef cortex.

> The adrenal origin of some sex hormones is supported further by the observation that the urine of castrates still contains sex hormones. These adrenocorticoids of the sex hormone type cause retention of nitrogen (a protein anabolic effect), phosphorus, potassium, sodium, and chloride."

It has been thought that the outer portion of the zona fasciculata is most active in the secretion of the cortical hormones.[5] Buonocore,[10] however, assigns definite secretions to each of the three main zones. According to his classification, desoxycorticosterone is associated with the zona glomerulosa, and a deficiency is manifested by vomiting, dehydration, fall in the alkaline reserve, and other metabolic changes. This is often accompanied by pallor, anorexia, asthenia, profuse sweating, and disturbances of respiration. Hyperfunction of this zone (excess desoxycorticosterone) would result in edema, hypertension, and left ventricular insufficiency. The fascicular zone produces secretions which control carbohydrate metabolism, and deficiencies are shown by vomiting, hypoglycemic crises, and convulsions, whereas hyperfunction leads to the classical syndrome of Cushing. Hypofunction of the zona reticularis (deficiency of androgens) produces the clinical picture of male pseudohermaphrodism, and hyperfunction is clinically manifested as female pseudohermaphrodism—the recognized virilism syndrome.

There is general agreement as to the classification of the cortical hormones into three main categories based on the physiologic effects of these steroids. Regarding the fetal cortex and its role, however, there is much more controversy. Its histologic appearance during fetal life and its sudden and complete involution following birth would lead one to suspect that it had a very definite and important function.

Goldzieher,[11] some years ago, observed that this involution did not take place in the cyanotic infant with congenital heart disease, and he presented the theory that the fetal cortex is pecularily adapted to facilitate the transport of oxygen to the tissues.

> An explanation [of the sudden involution of the fetal zone] is offered by the fact that the saturation with and the tension of oxygen in the fetal blood is considerably lower than in the maternal blood. The task of transporting oxygen to the tissues is facilitated by the increased number of red cells, but the utilization of "low tension" oxygen requires a specialized mechanism in which the cortical hormone plays a significant role. With the onset of respiration the oxygen supply is increased and the oxygen saturation of the blood reaches a point at which the demand for increased amounts of cortical hormone no longer exists. Hence, the precipitate involution of the superfluous fetal tissue after birth.

This theory has been refuted by Lanman[12] and others who found no delayed involution of the fetal zone in their study of seventeen infants dying of cyanotic congenital heart disease. Lanman says, however:

> The fetal zone, judging by its histological appearance and association with the adrenal gland, is presumably an endocrine organ. If size is an indication of activity it is also an active one, since the adrenal of the newborn is relatively 20 times larger than that of the adult. The fetal zone must perform its function primarily in the fetus, as it is present in an intact form exclusively during fetal life. . . . There are a few indications of a function related to that of the adult adrenal cortex. The tissue arises embryologically from the same site, though it is histologically distinct from the beginning. It also has a high concentration of ascorbic acid—as does the adult cortex—and is said to stain heavily for fat during fetal life.

Landing[13] observed abnormalities in size of the fetal cortex and noted that this zone was often small or absent in major brain malformations and large in cases of fetal asphyxia with edema. He points out that the connection of fetal edema with large fetal cortex, and polyhydramnios (reflecting fetal diuresis) with small fetal cortex might indicate that a salt- or water-retaining or antidiuretic, hormone is produced by this zone. A large fetal cortex has been found also in some cases of congenital syphilis and kwashiorkor, the malignant malnutrition of South African Negro infants.

Deane[14] suggests that

> . . . if the fetal adrenal is exposed to and is responsive to maternal corticotrophic hormone, the loss of this could account for the great involution of the gland at birth, which persists until the pituitary of the newborn has attained the capacity to secrete higher amounts of ACTH.

She also suggests the following hypothesis:

Investigation of the adrenals as the possible source of vital secretions began in 1855 when Addison noticed a connection between certain anatomic abnormalities of the gland and the clinical syndrome which now bears his name

The glomerulosa, before and after birth, is an actively secreting zone. . . . Prenatally, the cells in the fasciculata and reticularis are also secretory. After birth, in contrast to what occurs in the glomerulosa, the cells in the inner two zones shrink and the lipid droplets become enlarged. These alterations may be interpreted to indicate that after birth the cells of the fasciculata secrete at a somewhat slower rate than prenatally and those of the reticularis decline still further in secretory activity . . . that the glomerulosa secretes a mineralocorticoid, possibly aldosterone, and that its control is not solely through the pituitary. The fasciculata, completely under pituitary control, probably secretes the glucocorticoids.

The greater part of the knowledge regarding the adrenals has been gained through animal experimentation. It was from the results of such experimentation, begun in 1936, that Selye[15] developed his theory of the general adaptation syndrome (G-A-S). This theory, as first presented, has been proved invalid on many counts and is constantly being modified by its original proponent as new evidence becomes available. It has served, however, to direct thinking and research along lines which have proved to be very productive.

According to the original concept of the general adaptation syndrome, any form of stress, whether trauma, infection, extremes of temperature, physical exertion, or emotional strain, produces a specific reaction which is different for each stimulus, and also a stereotyped response which is common to all stimuli. The common denominator in all of these situations was that a state of biologic stress was produced within the body.[16]

Selye himself describes the G-A-S as consisting of three stages: (1) the "call to arms" or alarm reaction, (2) the stage of resistance, and (3) the stage of exhaustion. During the alarm reaction there are adrenocortical enlargement with histologic signs of hyperactivity, thymicolymphatic involution, eosinopenia, lymphopenia, polynucleosis, gastrointestinal ulcers, and often other manifestations of damage or shock.[17] During the second stage, ascorbic acid, cholesterol, lipids, and other storage products, which had been discharged by the cortical cells during the initial stage, are seen to reappear. During this time resistance to the specific stress is at a maximum; other types of stress, however, are met with lowered resistance. The histologic changes during the stage of exhaustion resemble those of the alarm reaction, but degenerative phenomena frequently occur and the organism loses its acquired adaptation.[15,17]

The medulla also takes part in the "call to arms" by immediately pouring into the blood stream an increased amount of epinephrine in response to stimulation through the sympathetic nervous system. The original concept, however, of initiation of the stress response by discharge of epinephrine from the medulla, which stimulated the anterior pituitary to increased production of adrenocorticotropic hormone and resulted in increased secretion of corticosteroids, has been largely replaced by a modified concept including the theory of dual regulation. Selye and Rosch[16] have summarized this newer concept thus: The stressor, such as trauma, infection, or burn, acting on the targets, produces a stimulus which causes the anterior pituitary to produce corticotropin; under certain circumstances it may also result in discharge of somatotropin. The nature of this first mediator between the directly injured organ and the anterior pituitary is still not known.

According to Forsham,[6] the adrenocorticotropic hormone (ACTH) is produced in the anterior lobe of the pituitary following stimulation by a humoral substance from the anterior hypothalamus. Secretory activity of this hypothalamic center is controlled by impulses from higher nervous centers in response to both physical and emotional stress. Harris[18] has expanded this concept to include a "feed-back mechanism" whereby adrenal cortical hormones and perhaps even epinephrine more probably act on a hypothalamic region than directly on the pituitary gland itself. Fortier[19] suggests that there is dual regulation of ACTH release, one entirely humoral in response to systemic stress stimuli and the other regulation probably neurohumoral, mediated by the hypothalamo-hypophyseal neurovascular pathway and coming into play under the influence of nervous or emotional stimuli.

Guillemin,[20] in interpreting the results of experiments with rats in which pharmacologic antagonists were used, states that epinephrine, norepinephrine, acetylcholine, and histamine were not indispensable links in the activation by neurotropic stress of the hypothalamic-pituitary axis, and they were not the only agents which could produce stimulation of the pituitary during systemic stress.

It is well known that insufficiency or lack of the adrenal cortical hormones can result in the clinical syndrome of weakness, anorexia, nausea and vomiting, diarrhea, loss of weight, inhibition of growth, fatigue, muscular asthenia, and greatly decreased resistance to stress. This is accompanied by laboratory findings of decreased serum glucose, sodium, and chlorides and increased serum potassium. These findings are thought to be the result of the inability of the renal tubules, in the absence of cortical hormones, to reabsorb sodium and chloride, and to excrete potassium in sufficient amounts to maintain normal electrolyte balance. This effect on the electrolyte balance is shared to some extent by all the known steroids produced by the cortex, but the chief control is exerted by desoxycorticosterone.[6]

It is quite generally agreed that cortisone and the other S hormones cause elevation of blood glucose, increase in gluconeogenesis with an increase in protein catabolism (or through a protein antianabolic effect),[21] increase in fat metabolism and mobilization of depot fat, decrease in carbohydrate oxidation, decrease in tubular reabsorption of urates, dissolution of lymphocytes, and involution of lymphatic tissues.[5,7]

The inhibitory effect of cortisone or hydrocortisone on inflammatory processes has been emphasized by many investigators. Forsham[22] envisions these agents as surrounding the cells with a protective covering which immunizes them to the usual deleterious effects of antigen-antibody complexes. He goes on to say, however, that this protection does not extend to outright toxins such as that of diphtheria. He also points out that

. . . the sequence of the cellular reaction to an inflammatory lesion is changed rather drastically from a preponderance of lymphocytes and fibroblasts to one of active macrophages and very few fibroblasts. Thus, the usually accepted barrier to the spread of micro-organisms is definitely removed, and the path is opened to the spread of organisms of low virulence in the form of superinfections.

Other investigators have found that cortisone depresses the phagocytic and regenerative activity of cells of the reticuloendothelial system[23] and interferes with the therapeutic effect of antibiotics.[24] However, the role of the adrenal cortex in the production of immunity through the formation and mobilization of antibodies is still undetermined.

Probably one of the greatest hindrances to progress in the study of the adrenals is lack of an adequate

method for measuring and evaluating cortical secretions. This is particularly true when it comes to the study of the human adrenal. One of the most readily available measurements of adrenal function, and therefore the one which probably has been most widely used, is the eosinophil count. Since this test is based on the response of the adrenal cortex to stimulation from the pituitary (or ACTH), it is predicated upon a well-developed pituitary-adrenal axis, which does not always obtain. The same criticism holds true for methods employing the urinary excretion of metabolic products of adrenal steroids, for in this case normal renal function is presumed.

Many physicians are understandably cautious in their approach to corticotropin and corticosterone

Other criteria which have been used include estimation of serum corticoid levels, blood glucose and electrolyte levels, electrolyte values of sweat and saliva, distribution and size of lipid globules and amount of depletion of ascorbic acid in the adrenal cortex, and determination of adrenal weight. All of these methods have serious limitations.

Symington and his associates[25] made an extensive study in which the factors of alteration in cell type, presence of degenerative changes in the cortical cells, distribution of ribonucleic acid and cytoplasmic enzymes, and distribution and character of the cortical lipids were evaluated as criteria for cortical activity. They classified cortical reaction to stress into three main patterns: (1) the "normal" lipid-laden adrenal, which they found only when the patient was unconscious during the period of stress, (2) focal lipid depletion, which is thought to be the normal response to stress found in both children and adults with slight modifications, and (3) extreme diffuse lipid depletion, occurring in severe stress, and characterized by definite cellular changes throughout the whole extent of the cortex, which was found to be rich in ribonucleic acid and, to some extent, in alkaline and acid phosphatase. The suggestion has been made that the focal areas of lipid depletion found during periods of slight or moderate stress may be interpreted as areas of activity and that the number and size of these focal areas are influenced by the intensity of the stress.

Considering what is definitely known concerning the functions of the adrenals and keeping in mind the plausible theories which have been proposed, these questions recur, "How much of this applies to the infant and child? What *is* the role of the adrenals during stress in infancy and childhood?" These questions have been asked many times, and a few investigators have tried to answer them.

Parmelee[26] has mentioned birth trauma as the first stress to which the infant is subjected. The position of the adrenals

> . . . exposes them to pressure and traction when the thorax is compressed excessively during birth. It may well be that this mechanism combined with passive congestion, especially in the presence of anoxia, accounts for adrenal hemorrhage as an immediate effect of the birth process. . . . Some infants with adrenal hemorrhage of lesser severity will survive the neonatal period and later show symptoms of hypofunction of the adrenal cortex.

The similarity of the symptoms of summer diarrhea in infants and the effects of hot, sultry weather on addisonian patients was noticed by Goldzieher.[1] He carries the analogy beyond the similarity of salt and water metabolism and points out that the inability to absorb carbohydrate and fat from the intestines is conspicuous in both conditions. The morbidity and mortality of small infants with upper respiratory infections also resembles the decrease in resistance of the addisonian patient. He states that true Addison's disease is rare in children, but names pseudopneumonia, the celiac syndrome, and malnutritional marasmus as diseases of infancy apparently related to adrenal insufficiency.

The suddenness with which the infant becomes dehydrated and develops hyperpyrexia is accepted by many as a manifestation of insufficiency of the adrenal cortex. The sequence of events probably begins with a disturbance of the water and electrolyte balance, due to insufficient cortical hormones, which quickly develops into dehydration. With subsequent hemoconcentration, increase in viscosity of the blood, and slowing of the blood stream through the capillary network of the skin, there is interference with the peripheral heat regulatory mechanism, and increased temperature is the result.

Goldzieher[27] speaks of the precarious equilibrium of the homeostatic mechanisms in the infant and young child and refers to a state of latent cortical insufficiency.

> In the normal child, the progress of involution does not exceed the physiologic limit, yet the profoundly altered cortical tissue is hardly capable of maintaining more than a state of marginal sufficiency, which may be adequate under favorable conditions, but which will prove unable to cope with suddenly increased demands for the cortical hormone. It is justifiably postulated, even on the strength of the morphological evidence alone, that a state of latent cortical insufficiency exists in the infant from the time of birth until the function of the remaining cortex becomes stabilized after completion of the involution. It is most probable that Nature provides, in the meantime, other temporary adjustments, which safeguard survival and simultaneously ease the burden of the cortex in the maintenance of tissue metabolism. Yet the functional equilibrium attained by these adaptations remains precarious at best.
>
> The period of latent adrenal insufficiency extends at least over the first six months of life. A limitation of cortical reserve power of lesser degree seems to prevail for a longer period, which may last to the fifth or sixth year of life. This period of the child's existence is characterized by a peculiar response to its environment and by marked susceptibility to characteristic pathological manifestations.

This concept of cortical insufficiency during infancy and childhood has been questioned by many investigators, and reports of experiments and observations are confusingly contradictory.

Some studies, using the fall in circulating eosinophils as the index, have shown that practically all term infants respond with a drop of more than 40 per cent, but that in prematurity the amount of decrease is lessened.[28] Other workers have found no significant change in the eosinophil count in the newborn under stress situations, perhaps indicating failure of response to stress.[4]

The measurements of Venning, Pattee, and Beck[5] reveal adequate quantities of glucogenic corticoids in newborn infants. Still other determinations, using levels of sodium and other elements of the blood as in-

dexes of adrenal cortical function, have shown a rather stable and constant level existing during the neonatal period.[29]

Many reports indicate that, during periods of stress such as infections or surgery, the infant adrenal responds with secretions of large amounts of corticosteroids. Emotional stress also has been found to increase the corticosteroid level in older children. The blood corticosteroid levels in children dying of meningococcemia, as well as those with uneventful meningitis, have been found in many cases to be very high.[30] Talbot,[31] in studying several sick infants, found the urinary corticosteroid levels to be remarkably high in absolute amounts and, even more so, when expressed on a square-meter-per-day basis, sometimes being greater than those seen in Cushing's syndrome.

Klein[30] has summarized well the findings of numerous investigations of infants under nonstress situations:

> There is a period of diminished adrenal activity during the first week of life. At birth, the blood corticoids are at adult levels; by the third day they drop to very low values and then slowly rise again to levels seen in adults. The 17-ketosteroids are likewise high during the newborn period, but disappear almost entirely during the first week of life. All the available data support the premise that following birth there is a period of lessened adrenal function and lowered responsiveness to ACTH.

A segmental or selective insufficiency has been shown to exist in adult patients, in which condition the clinical syndrome may be that of Addison's disease, but laboratory reports may show a normal glucose tolerance curve and normal range of glucocorticoids.[5] This type of insufficiency occurs also in congenital adrenal hyperplasia, in which the synthesis of 17-hydroxycorticosteroids is impaired and there is overproduction of adrenal androgens. It has been suggested that this imbalance may also exist in rheumatic fever[32] and could possibly account for many unexplained syndromes of infancy and childhood.

The close relationship between the various hormones of the body is worth noting. Stress stimuli seem to excite the adrenal cortex and inhibit the thyroid gland. This may indicate reciprocal relationship between the secretion of adrenocorticotropic hormone and thyrotropic hormone. Two forms of stress, however, cold and injection of cortisone, seem to change the rates of secretion of these two hormones in the same direction.[33] The inhibition of adrenal cortical growth in hypopituitarism is well known, as is also atrophy of the adrenals following hypophysectomy. Corticoids, follicoids, and testoids have all been shown to cause atrophy of the thymus.[16] Sayers[34] suggests also that optimal response of the adrenal cortex to corticotropin, and of other tissues to cortical hormone, depends on the presence of a normal titer of thyroid hormone.

Forsham[35] has suggested that unresponsiveness of the end-organ may be the real basis for what often appears to be hormonal insufficiency. It is possible that the infant may be capable of secreting adequate amounts of hormone, and indeed may do so, but that the pathways for response are not yet fully developed.[4] It may well be, as suggested by Jailer,[28] that myelinization of the tracts from the hypothalamus to the pituitary must occur before the infant can respond to stress.

In the light of what is known and accepted regarding the stages of development of the adrenal in childhood and the probable functioning of the gland, is there not justification in accepting as highly probable the concept of latent insufficiency of the adrenal? Is it not possible that some of the fulminating infections, which all too often snuff out young lives, are so deadly because of accompanying adrenal crises brought on by the stress of the infection? Graham[36] has attributed sudden death in young adults with fatty livers to probable hypoglycemia. May not some of the sudden deaths in infancy be ascribed to this same cause and be the result of deficiency of the adrenal glucocorticoids? Could it not be possible that the unexplained acute abdominal pain accompanying many childhood illnesses may be an indication of acute adrenal insufficiency?

Whether the pediatrician accepts the theory of latent cortical insufficiency, or points to the unresponsiveness of the end-organ as the basic problem, or believes that some as-yet-unthought-of mechanism is at work, he is still confronted with a definite challenge each time he is called upon to care for one of these tiny patients. What can he do to help them safely through a period of stress which might prove fatal?

Probably the best aid a physician can have in the diagnosis of any condition is a high degree of suspicion. If he keeps in mind that the hormonal balance of the infant and young child is, at best, precarious and is constantly on the alert for signs of imbalance, there will undoubtedly be many instances in which judicious use of the hormonal preparations now available may be lifesaving.

During the last few years, corticotropin and cortisone have been used in a wide variety of diseases. Palliation has been achieved in many of the chronic diseases and those characterized by recurrences, but the effects are generally not maintained when therapy is discontinued.[37]

Many physicians are understandably cautious in their approach to these hormonal preparations. When using them as therapy, one must weigh such beneficial effects as increased resistance to stress, cellular protection against toxins, and possible increase in antibody mobilization against the deleterious effects of breakdown of tissue barriers to the spread of infection and decrease in therapeutic efficacy of antibiotics.

Rheumatic fever has been treated on the theoretical basis of having adrenocortical insufficiency as the underlying factor. The logical rationale for present-day ACTH and cortisone therapy in rheumatic fever is presented by Kelley:[32]

Despite the great strides made . . . in the knowledge of adrenal physiology, there remain numerous unsolved enigmas

> During the initial week of illness, the steroid concentrations in children with rheumatic fever were higher than the mean control value. Thereafter, the levels decreased progressively. After the fourth week of illness, not a single patient had a steroid concentration as high as the normal mean value; low values have been found several years after the disappearance of all evidence of rheumatic activity. The variations from normal are statistically significant . . .

There is definite evidence of abnormal pituitary-adrenal cortical function in rheumatic fever, similar to that which exists in Addison's disease, congenital adrenal hyperplasia, and adrenalectomized subjects. This provides rationale for cortisone therapy in this disease. Since rheumatic fever patients increase their serum steroid concentration in response to stimulation by exogenous ACTH, and during the acute phase of the disease by response to endogenous ACTH stimulation, the adrenal insufficiency is a relative rather than an absolute one. This makes the use of ACTH therapy as logical as the use of cortisone.

Kelley explains the beneficial effects of salicylate therapy as possibly being mediated through the pituitary-adrenal system, producing increased utilization of the steroids, and concludes with the statement,

Whether there is increased utilization or adrenal insufficiency, the homeostatic mechanisms in these patients are such that the adrenals are not able to maintain normal circulating concentrations of the adrenal steroids. Response is not adequate to meet the situation with which the adrenal is faced.

Experiments have shown that the life of adrenalectomized animals can be greatly prolonged by the administration of sodium salts. Diets high in sodium and calories and low in potassium have proved very beneficial in cases of Addison's disease[5] and might well prove of value when the patient is a child under the stress of infection or trauma.

Despite the great strides made during the last decade in the knowledge of adrenal physiology, there still remain numerous unsolved enigmas. Each new discovery seems to open up wider fields for research and with each report of new investigations comes the expressed or implicit admission, "Much yet needs to be learned."

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Infancy and childhood

Infancy and childhood are, in certain respects, the most important and interesting periods of life. To the physiologist they are especially interesting, because they are the periods of development and of greatest functional activity; to the pathologist because in them many diseases occur which are rarely or never observed in the other periods, or which present in these periods peculiar features; to the physician and vital statistician, because in them the greatest amount of sickness and the largest number of deaths occur . . . The causes of this great mortality of infants and children, and the means of diminishing it, deserve careful attention.—J. Lewis Smith, M.D., "A Treatise on the Medical and Surgical Diseases of Childhood," 1896.

Review of the care of the PREMATURE INFANT*

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ANY DISCUSSION OF care of the premature infant could well begin or end with the statement of Dunham[1] that we actually know better what not to do than what to do. What is the cause of this problem? Why prematurity? It is well known that there is no one answer. However, I should like to list the known causes of prematurity and then to dwell upon the unknown causes. According to Clifford[2] 8 to 13 per cent are due to multiple births, 10 per cent to maternal toxemia, 10 per cent to maternal bleeding, and 4 per cent to malformation or erythroblastosis; 60 to 70 per cent are spontaneous with no known complication.

Even with the high quality of present-day prenatal care, it has been shown that 30,000 more babies could be saved every year if public and professional cooperation were available to put into effect the best that is known of prenatal and postnatal care. For instance, termination of multiple pregnancy usually occurs about 3 weeks ahead of time. Knowing this, why not prepare for the event? The mother, of course, requires special prenatal attention, nutrition, and rest. The obstetrician caring for this woman should consult with the pediatrician and, as a team, they should decide upon her care. At delivery, the second or last infant is usually weaker, and preparation for this is most important as is gentleness at delivery.

Toxemia is less common each year, probably because of better prenatal care, with stress on regular examinations that result in finding the symptoms of toxemia early, and restriction of salt, attention to diet, and rest, as indicated. Patients with toxemia are more willing to be hospitalized, and hospitals are more willing to accept them, than was previously the case. Having a pregnant mother rest in the hospital for several weeks is much less expensive than is nursery care for 2 to 3 months for the premature baby.

The bleeding patient can avail herself of hospital care, with blood banks for support, which allows for conservative management and prolongation of the infant's intrauterine existence. Every week of delayed delivery means a weight increase of 4 to 8 ounces for the baby; and with each 8-ounce gain, the premature mortality is cut in half.[2]

Possible erythroblastosis is rarely an indication for terminating pregnancy, since exchange transfusion can be fairly easily performed after birth. The incidence of death from kernicterus is favorably overbalanced by birth of a more mature infant.

It is with the 60 to 70 per cent of spontaneous, uncomplicated premature births that this paper is concerned. We, as pediatricians and specifically as osteopathic pediatricians, should study these. I believe it would be extremely interesting to know the prenatal management of the mothers of these infants, particularly with regard to osteopathic management. Questions that arise in relation to the use of manipulative therapy are:

1. Has regular osteopathic manipulative therapy effected a reduction in the incidence of premature delivery in this group?
2. Is there a consistent maternal spinal finding pattern in these cases of prematurity?
3. Is there a consistent spinal pattern found in infants born prematurely?

I raise these points because prematurity is the greatest cause of infant mortality. It is true that there has been a drop in neonatal mortality from 32.5 in 1935 to 20 per 1,000 in 1951, but it is also believed that a rate of 13 per 1,000 could be achieved.

As used in this discussion, the term "premature infant" refers to one weighing less than 2,500 grams (5.5 lbs.) at birth, regardless of the period of gestation. The incidence of prematurity in the United States is not known exactly, but it is estimated to be about 7 per cent of the total live births.[3] There are approximately 3,000,000 live births a year in this country, which would mean that 210,000 premature infants are born annually. Approximately 70 per cent of these weigh between 2,000 and 2,500 grams; 18 per cent weigh from 1,500 to 2,000 grams; 7 per cent weigh between 1,000 and 1,500 grams; and 5 per cent weigh less than 1,000 grams.

The problem of care of the premature infant can be divided into five distinct phases: (1) the prenatal period, (2) birth, (3) emergency phase—from birth to about 7 days, (4) alimentary phase—the third to the twentieth day, and (5) the growth phase.

Prenatal period

The most important aspects of the prenatal period are cooperation between the patient and her obstetrician and cooperation between the obstetrician and the pediatrician.

Birth

A physician trained in the care of the newborn infant should attend the delivery of all suspected premature infants. This rule is a good one and is in effect at the Massachusetts Osteopathic Hospital and in most other hospitals today. The physician should see that the incubator is preheated and in readiness and that suction and oxygen are also ready for use.

The obstetric responsibility is great. The obstetrician must do his best to prolong pregnancy, but if premature labor commences, his main consideration is the avoidance of fetal asphyxia if neonatal mortality is to be prevented. There are many ways that asphyxia may be produced before birth:[4]

1. Interference with placental circulation
   a. Placenta previa
   b. Abruptio placentae
   c. Compression of the umbilical cord

*Presented at the Annual Meeting of the American College of Osteopathic Pediatricians, New York City, July 14, 1956.

2. Severe maternal anemia
3. Maternal low blood pressure
4. Excessive maternal sedation or anesthesia accidents causing anoxia in utero.

This last results in a high immediate mortality rate or in permanent cerebral damage and can be prevented only by rapid delivery. Sedatives and anesthetics of a general type can be given to the mother only at the expense of the infant. The margin of tolerance of the premature baby is even less than that of the full-term infant; therefore, it is best to give no sedatives or, next best, to keep them at an absolute minimum. Under no circumstances should cyclopropane be used as it produces fetal anoxia.

In cesarean section the infant usually weighs more than 5.5 pounds, but his actions are those of a smaller infant. The infant is certainly immature; otherwise, birth would have occurred before section was performed. I believe all babies delivered by section should have the best of pediatric attention. The pediatrician should be at the section prepared to receive the infant as soon as it is delivered. Most of these babies need aspiration of the stomach as well as of the oropharynx. They should be put on the premature infant service and fed as if they weighed less than 5.2 pounds. An infant weighing 6 pounds when delivered by section 2 weeks before term would have weighed about 7 pounds at normal delivery.

Delivery is best done from below by means of episiotomy and forceps. Delivery by breech extraction is accompanied by the highest mortality and requires expert obstetric judgement. Delivery from above or below should be followed by placing the infant below the level of the placenta in order to allow blood to flow to the infant by gravity and a slower return; the cord should not be cut until pulsation ceases. Most physicians also give the mother 50 to 100 per cent oxygen during the last phase of the second stage of labor; this seems to assist fetal circulation and may ward off fetal anoxia.

At least 1 inch, and preferably 2 inches, of the cord should remain after it is cut. If erythroblastosis is suspected, the umbilical vein is then easily exposed, and polyethylene tubing can be inserted into the vein by means of an Allen needle or cannula. This permits easy exchange transfusion or blood studies if necessary.

Emergency phase

An inadequate oxygen supply to the brain will cause irreversible injury in only a few minutes. Frequently associated with anoxia in the small premature baby is the development of intraventricular hemorrhage originating from the choroid plexus or vena terminalis of the lateral ventricles. These are the cases in which the infant does well for 2 or 3 days and then develops periods of profound apnea and dies. No treatment is known.

The pediatrician present at delivery must be ready to begin resuscitation, which means that he must provide an open airway, minimize shock, and get oxygen to the lungs. The premature baby is in a state of shock. He may have a cerebral injury. He is partially anesthetized and may have aspirated mucus. He has been compared to an intoxicated man who has been struck by an automobile and knocked to the pavement. Both should be moved as little as possible and kept warm, and should have the mouth kept open and the throat cleaned.[4] Treating the premature in any other way shows ignorance of basic physiology and pathology. Resuscitation must not involve violent or vigorous manipulation.

Warmth is next in importance. The infant has been in a water bath for a few months at a temperature of about 100 F. A delivery room at 80 F. seems hot, but it is a 20-degree drop in temperature for the infant. He will lose body heat rapidly in this environment, and this results in more shock; hence the incubator or heated bed should be ready. Aspiration and oxygen therapy can be carried out in these.

If postural drainage is utilized, most premature babies need only mild aspiration of the oropharynx to provide an airway. This drainage position should be a tilt of only about 15 degrees so as not to increase possible cerebral hemorrhage. Aspiration of the stomach should be attempted gently with a No. 8 French catheter attached to a vacuum line. At our hospital we use controlled vacuum pressure of not more than 40 cm. of water, believing that, with pressure greater than this, damage may result to the gastric or esophageal mucosa. Aspiration of the trachea is usually necessary, but when performed, it should be by direct observation with a laryngoscope, if possible, to reduce trauma. Foreign material in the trachea is extremely rare.

Delayed respiration is not cause for alarm, provided the infant's reflexes are at least partially intact and there is some pink coloring to the skin. At the Massachusetts Osteopathic Hospital treatment is based on the maturity of the infant and the extent of apnea. For most large premature babies a face mask with oxygen and a rebreathing bag are all that are necessary, as the infant is already making inspirational efforts; if he is not, rhythmic pressure on the bag helps. Smaller premature babies will need more assistance—oxygen delivered under pressure or through a catheter. If respiration is delayed beyond 2 minutes, two gastric catheters are placed, and oxygen is given through one catheter and the other is used as an exhaust. Numerous resuscitative methods have been developed to produce positive or negative pressure, but we have never used them. Cole[4] claims that none of these devices have proved very satisfactory with premature infants. The mask and rebreathing bag are useful in this situation for smaller infants so long as the pressure does not exceed 30 mm. of mercury and the bag can be equipped with an automatic cutoff valve.

Various drugs have been used to stimulate respiratory efforts; among these are Coramine, alpha-lobeline and Metrazol. The effect of most has been brief stimulation followed by depression. However, it must be remembered that the trouble is, in most instances, in the lungs themselves, and that no amount of stimulation to the respiratory center will expand a collapsed lung. When the respiratory center has been depressed by drugs or anesthetics, caffeine sodium benzoate may be of value in small repeated doses of ¼ to ½ grain. Nalline, 2 to 3 mg. via the umbilical vein immediately after delivery, may also be of value in combating narcotic depression.[3]

At the Boston Lying-in Hospital, all injections in premature infants are given in the lower third of the anterior aspect of the thigh. There is sufficient tissue at this spot, and injection there requires less handling of the infant than injections in other areas. The area is not covered, it is clean, and it can be observed very easily for hematomas or inflammation. I use gentle upper dorsal extension with oxygen, and although the

mechanism is not entirely clear, weak respiratory efforts frequently follow this procedure. However, it has been shown that it is not until the gestational age of 28 weeks and a fetal weight of 1,000 grams that enough capillaries have penetrated the oxygen barrier of cuboidal cells lining the pulmonary tree to make oxygenation and continuous life possible.[2] This is an important consideration in abnormal pulmonary ventilation.

As soon as respiration has been established, the infant is no longer handled; he is placed with his head lowered, without clothing, and oxygen is used only as required. There is no general rule that can be given for oxygen therapy in the premature baby, and administration must, in my opinion, be governed by the needs of the infant. In our hospital we usually provide an atmosphere of 40 per cent moist oxygen for any infant who exhibits an anoxic tendency. If this does not prevent cyanosis, the amount of oxygen is raised to 50 to 60 per cent, depending on the concentration necessary to prevent cyanosis.

The premature infant exhibits evidence of anoxia as a usual, rather than as an unusual, circumstance. Anoxia is the premature's greatest enemy in the first few days as it produces a chain of events affecting every part of the body.[2] The results of anoxia are congestion, edema, hemorrhage, and tissue death. The clinical symptoms of this will depend upon the organs involved.

The baby is placed in an incubator, and the temperature is regulated according to the infant's body temperature. As soon as the infant's axillary temperature is stabilized at 97 or 98 F., he is removed to a regular crib. Alevaire has been advocated by many authors, to be used as a moistening agent and attached as a nebulizer to the oxygen flow of the incubator. At Massachusetts Osteopathic Hospital we have not been convinced of the usefulness of Alevaire in the prevention or treatment of respiratory difficulty in general, but do use it in some cases for about the first 24 hours.

The greatest postnatal problem of premature infants is the development of a hyaline membrane. This has been reported to cause the death of 60 per cent of premature infants dying between 4 hours and 4 days after birth.[2] The lesion may be found alone or with other serious disorders. Since most babies recover, however, it is difficult to evaluate therapy. They need oxygen and water vapor, but other wetting agents, according to some, have no effect.[2] Antibiotics are recommended.[2] Extrauterine respiration of at least 1½ hours appears necessary for development of these membranes.[5] Anoxia is probably the cause of hyaline membranes as it leads to capillary permeability with exudation into the alveoli and to aspiration of meconium-containing amniotic fluid, which in itself increases capillary exudation from the alveolar septa. Forced respirations result as the exudate forms, but fluid is absorbed as the exudate dries, and a hard membrane results. Clotted terminal bronchioles create atelectasis, and anoxia increases until asphyxiation occurs. If the changes are minimal, and in most cases they are, there is time for a leukocytic response and resolution of the membrane; thus a reaction is reestablished and recovery takes place.

Other problems in the emergency phase may be intracranial hemorrhage, subarachnoid hemorrhage, and congenital malformations.

Infections such as epidemic diarrhea should be considered as preventable or controllable, if strict nursing technic is used. Since the premature infant is prone to infection, the following recommendations are made:

1. Isolation of the nursery itself.
2. Terminal sterilization of formulas.
3. Isolation of infants by a good incubator or crib technic.
4. Prophylactic aqueous penicillin, 50,000 units intravenously every 6 to 8 hours for 7 days, on indication.
5. Sodium sulfadiazine, 40 mg. per pound intramuscularly in 24 hours for 7 days.

Hemorrhagic diseases may constitute complications; these include erythroblastosis, sepsis, anoxia, and trauma. A true defect in blood coagulation may occur, such as absence of fibrinogen. This requires transfusions. There is much conjecture about administration of vitamin K in hemorrhagic conditions. However, until a solution is reached I shall continue to give 1 ampule to the infant at birth and daily for 3 days if bleeding is feared.

Alimentary phase

It is customary to withhold foods and fluids from premature babies for periods varying from 24 to 72 hours or longer. The smaller the baby, the longer the delay. The baby, particularly the small one, has enough trouble for the first 2 or 3 days without worrying about feeding. The high humidity allows for some fluid absorption. At the Massachusetts Osteopathic Hospital we follow closely the routine of the Boston Lying-in Hospital except in regard to breast milk. We feed the baby dry skimmed milk. Boston Lying-in Hospital has a mothers' milk center, and therefore has no problem. Authorities seem to be about equally divided on the subject of breast milk or formula feeding.

Most hospitals start feedings by gavage if there is any question of the infant's ability to suckle or swallow. As the infant shows evidence of wanting to suckle or swallow, he is permitted to do so. Total fluids are maintained by clysis if vomiting occurs. Clysis consists of .45 per cent saline and 2½ per cent glucose in water solution. The calorie intake is maintained at 60 calories per pound after 5 days. Laboratory control by hematocrit and microchemical electrolyte tests is helpful in difficult cases but is not usually necessary. Total fluid should be provided as soon as possible by suckling and swallowing as the open gullet is the best means of assuring electrolyte and fluid balance.

Growth phase

At the end of the period of initial weight loss, when a leveling or gain occurs, the calorie intake is adjusted to allow for a steady gain of about 1 ounce per day. Iron is usually added at this time in the form of ferrous gluconate or sulfate.

A survey of the home to which the infant will go on discharge should be made by someone from a public health and welfare association or a person experienced in such an evaluation. If home conditions are good the infant may be discharged when he weighs 5 pounds; if poor, the infant should weigh more than 5.2 pounds. Some states provide financial support for premature infants while they are hospitalized. In Massachusetts, the Public Welfare Department pays the expenses of all premature babies weighing less than 5 pounds at birth if the family is unable to do so.

Studies of premature babies show that those

weighing more than 4 pounds develop about the same problems as heavier infants. Those under 4 pounds in weight, however, show a greater incidence of cerebral spasm, delayed development, retrolental fibroplasia, bilateral cataracts, and emotional instability.[6]

Conclusions

Pediatricians have an opportunity to save many premature infants by conscientious, sensible care in the first 72 hours, and more may be saved by good obstetric and osteopathic management. Warmth, immediate respiratory tract drainage, moist atmosphere, and oxygen by demand are the first considerations. Gavage feeding by means of an indwelling polyethylene catheter and proper attention to total fluids by laboratory means or careful clinical evaluation constitute the next consideration. Prophylactic administration of antibiotics with careful injection into the anterior thigh should be thoughtfully considered. Exchange transfusions should be immediately available in cases of hemolytic diseases. Possibilities for proper home care should be evaluated and the family taught how to care for the infant.

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PEDIATRIC ANORECTAL disorders*

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DISORDERS OF THE anorectal region are often overlooked as a possible cause of crying in the infant. The physician is too quick to attribute the crying to colic, to change the formula, and to accuse the mother of being nervous. The anorectal region is a richly innervated area in which disorders may give rise to exquisite local pain and reflex systemic effects. The need for treatment is frequently urgent, and results are often gratifying.

Anatomy

A brief consideration of the applied anatomy pertinent to anorectal disease in the infant will be helpful.

The anal canal begins at the anorectal line above and ends at the anal orifice. It is the terminal portion of the large intestine and in infancy is directed downward in a straight line. It is a continuation of the rectal tube and is rather insecurely attached to the almost straight sacrum and coccyx. This anatomic characteristic appears to play an important role in the lateral distribution of fissures and fistulas and in the occurrence of procidentia in infants up to 2 years of age.

The sacral and rectal relationships change as the infant grows into childhood, the sacrum assuming a deep anterior concavity. The rectum is then drawn posteriorly and assumes an almost right-angle relationship with the anal canal which at this time runs in a more posterior direction. This anatomic development gives better support to the rectum and directs the area of trauma by the oncoming stool to a point in the posterior arc of the anal canal. It explains the reduced incidence of procidentia in childhood and the fact that fissures and fistulas tend to occur more frequently in the posterior position rather than in the lateral position as seen in infancy.

The anal canal derives its muscular support from the levator ani and is surrounded by the superficial and deep external sphincters. The upper portion of the anal canal forms an irregular border by its union with the bases of the rectal columns of Morgagni. Stretched transversely between the bases of these adjacent columns are thin semilunar folds, known as the anal valves or crypts of Morgagni, which correspond to the number of columns present. The crypts are in direct communication with the deep intramuscular glands via the anal ducts, and these three structures play a very special part in the pathogenesis of anorectal disease. Infectious material from a soft or runny stool may enter an anal crypt and be funneled through the duct into the deep intramuscular glands. Because of their poor drainage these glands easily become infected, and the infection is spread directly to the perianal and perirectal tissues when the gland ruptures or indirectly to these areas via the blood vessels and lymphatics. Acute thrombotic hemorrhoids, anal fissure, papillitis, and abscess and fistula formation are some of the clinical entities which may ensue.

The anal papillae or papillae of Morgagni are small teatlike elevations situated at the bases of the columns of Morgagni along the anorectal line. Frequently these papillae become inflamed or undergo hypertrophy and cause sphincter irritability with consequent constipation.

The rectum is approximately five times the length of the anal canal. It communicates with the sigmoid colon at a point opposite the third sacral vertebra and in its descent lies in the concavity of the sacrum and coccyx. It then rests on the pelvic floor formed by the union of the two levator ani muscles. It ends at the anorectal line, which represents the junction of the rectum above with the anus below, and, embryologically, is the point of union between the hindgut and

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the proctodeum. The proctodeum is the point on the fetal perineum where the anal dimple invaginates to meet the gut tract, establishing communication with the exterior. An arrest in development of this process or failure of the anorectal plate to rupture properly will result in varying degrees of imperforate or stenotic anus.

The rectal mucosa is quite vascular and is a deeper red in the infant than in the adult. This congestion of the mucosa, particularly when the baby strains at stool, causing a slight prolapse, is frequently mistaken for hemorrhoids. The mucosal surface is somewhat irregular and is lined with columnar epithelium. It presents three transverse or oblique folds known as the valves of Houston. These valves are semilunar, or crescentic, in shape and extend for a short distance about the circumference of the rectum. They are situated from 3 to 5 cm. from each other and are supposed to lend a spiral motion to the fecal mass in its passage through the rectum.

The pelvirectal sphincter lies at the junction of the sigmoid colon and rectum. It serves as a restraining barrier for the storage of fecal material in the sigmoid, and when the feces pass into the rectum the sphincter permits the build-up of the necessary intrarectal pressure for the stimulation of the rectal reflex. The internal sphincter consists of a thickened ring from the involuntary circular fibers of the rectum at the anorectal junction. It is innervated by the autonomic nervous system. Its tonicity is decreased with parasympathetic stimulation and increased with sympathetic stimulation. The external sphincter is composed of voluntary striated muscle which enfolds the anal canal. It is innervated by fibers derived from the sacral plexus and is under voluntary control of the cerebrospinal system.

The venous return of blood from the rectum is of clinical importance since reactions secondary to anorectal disease such as thrombotic and septic emboli can be mediated through major venous tributaries to the liver, heart, and lung. The superior hemorrhoidal plexus drains the rectum and ultimately enters the splenoportal system. The inferior hemorrhoidal plexus originates below the anorectal line, draining the structures below the pelvic floor. This system ultimately enters the heart through the inferior vena cava.

Examination

The infant's inability to tell just where and how he hurts places the pediatrician in the veterinarian's dilemma. He must therefore make the most of whatever physical signs are presented, for in proctologic disease, these signs alone, without a history, will frequently offer a definitive diagnosis.

Of course, the history is of value. The mother will report that the baby cries shortly before, during, or after defecation, or she may report seeing blood or mucus in the stool. Diarrhea or constipation may be the presenting complaint, or a protrusion from the rectum may be the reason the infant is brought into the office. Or perhaps the complaint is simply that the baby is unhappy and irritable. The first group of complaints should lead directly to careful examination of the anorectal region. The latter complaint should suggest the need to rule out disease in this area as a source of chronic discomfort.

With the need for meticulous examination thus established, the following procedures are recommended: The infant most easily assumes the knee-chest position, and the buttocks are gently separated. The perianal skin is observed for discolorations, eruptions, inflammation, induration, scars, sinus openings, fissures, and evidence of external invaders such as worms and lice. The contour of the anal opening is noted, and gentle introduction of the tip of the little finger into the anus will cause the infant to strain and thereby will tend to evert prolapsing mucosa or polyps or to expel excretions such as mucus, pus, or blood. Inspection of the sacrococcygeal area, perineum, buttocks, and vulva should also be carried out at this time.

Gentle palpation of the above mentioned areas for evidence of subcutaneous tenderness or masses should precede monodigital examination of the rectum. It is well to remember that even in the young infant physical examination is incomplete without digital exploration of the rectum. The little finger should be well lubricated and slowly inserted into the anus. Gentle, steady pressure exerted against the sphincter will cause it to relax and facilitate entrance into the rectal canal. With the anterior and posterior aspects of the finger facing the lateral walls of the canal, sphincter spasm, tenderness, local temperature changes, and masses in the rectum or adjacent organs will be more easily elicited. Then by placing the other hand on the abdomen, bimanual examination of the rectopelvic area is carried out. Closer examination of the anus and perianal regions for the presence of deep localized areas of induration or suppurative processes is effected by a bidigital technic of palpation. The index finger is placed in the rectum, and the thumb of the same hand is placed on the perianal skin. Gentle palpation of the intervening tissues is then performed in a clockwise or counterclockwise direction until the entire perianal circumference is examined.

Visualization of the anal and rectal canals with adult instruments may be carried out without trauma in infants past 1 year of age if gentleness and care govern the procedure. For younger infants smaller endoscopic instruments have been designed. The use of the anoscope and proctoscope is certainly within the pediatrician's technical ken. Sigmoidoscopy, however, is a technic which requires special skill and is best left to the proctologist or abdominal surgeon. When painful lesions such as fissures or thrombotic hemorrhoids are suspected, anoproctoscopy should be carried out after topical anesthesia is produced with Tronothane jelly.

Of course, biopsy of all neoplastic lesions should be performed before institution of definitive therapy. However, this procedure should be carried out only by those who are experienced in the technic and are prepared to stop hemorrhage. Adenomatous lesions must be removed in their entirety, since piecemeal biopsy tends to favor their spread. Papanicolaou smear technics on excretions obtained from below and above a lesion in the rectum are now in the process of evaluation and hold promise of accuracy.[1]

In protracted diarrhea, culture of the stool for bacteria or parasites may be of value. Culture of a discharging fistula provides information for the rational choice of an antibiotic. The cutaneous Frei test is used when rectal strictures are encountered and, of course, one of the chemical tests for occult blood should be a routine office procedure. Chronic blood loss will be reflected by lowered hemoglobin, and early suppuration will be revealed by an increased erythrocyte sedimentation rate.

In Mentzer's[2] series of 147 pediatric proctologic cases, pain and bleeding were the most frequent pre-

senting complaints. Constipation or diarrhea was frequently associated and constituted an important part of the pathogenesis. Abrasions in the lining of the anal canal produced by a hard stool or by irritation of diarrhea make a ready portal for entrance of infecting bacteria into the deeper perianal structures.

The history most frequently elicited is that of a baby who cries during the act of defecation. This complaint should direct the pediatrician to the broad group of inflammatory lesions, and of these, fissure in ano will be the one most frequently found.

Fissure in ano

In the infant a fissure is usually produced when the anal skin is lacerated by the passage of a large, hard stool. The laceration thus produced usually heals promptly, but with repeated trauma, healing is prevented and a linear ulcer or fissure will form, most frequently on the lateral walls just distal to the mucocutaneous junction. When chronicity ensues, the fissure has a gray base, slightly indurated edges, often discharges small amounts of pus, and bleeds slightly with defecation. Sphincter spasm and the exquisite pain associated with bowel movements constitute both involuntary and voluntary causes of constipation. A troublesome fecal impaction frequently results when, in order to avoid the pain of fissure in ano, the patient resists the urge to defecate.

Conservative treatment will effect a cure in almost all cases of fissure in ano in infants. The stool should be softened with a 1 ounce warm, mineral oil retention enema twice daily. The application of a topical anesthetic-antibiotic ointment such as equal amounts of Tronothane jelly and Polysporin ointment will give comfort and promote healing. Painting the lesion once or twice at 3- to 4-day intervals with a silver nitrate stick is particularly helpful. If recovery has not taken place at the end of 1 week, gentle dilatation of the external sphincter should be performed by the mother twice daily.

Anal abscess

The next most frequently occurring inflammatory lesion seen in the infant group is anal abscess. The baby is irritable, cries frequently, sleeps poorly, and may resist sitting up. Examination will reveal localized involvement, usually on the lateral wall of the perianal area. The tissue is hot, indurated, and red, and varying degrees of fluctuation may be present, depending upon the stage of the abscess. The temperature is usually elevated, leukocytes are commonly increased to 16,000 to 25,000, and the erythrocyte sedimentation rate is usually elevated. If the crypts of Morgagni are examined at the anorectal line, associated cryptitis is frequently seen, for it is here that the abscess has its inception. The direction in which the infection spreads determines the site of the abscess. The anal abscess which involves the external intersphincteric area is the most frequent abscess in infants. The incidence of supralevator spread is higher in children and adults. If the infected crypt closes off, thereby preventing drainage into the anorectal canal, the abscess, following the line of least resistance, will rupture into an adjoining structure or establish communication with the exterior, and a fistula will be the result. The fistula is an organized channel with its internal opening at the crypt in the anorectal canal and its external opening discharging the products of infection to the exterior. If the fistula has burrowed and branched, the external openings will be numerous and the amount of contiguous tissue involved will be great.

The only treatment of anal abscess is immediate incision which is deep enough and long enough to insure thorough drainage. The physician should not temporize. Waiting for the abscess to point before incision, or hoping to abort it with antibiotics alone, is a dangerous mistake, for it is during this period of wishful thinking that fistulous connections may form between the abscess and adjacent areas other than the skin.

With the patient in the exaggerated lithotomy position, an anteroposterior incision is made over the maximum point of fluctuation as close to the anal opening as possible but lateral to the outermost fibers of the external sphincter which, in the infant, extends laterally from the anal slit for ½ inch. If at first no purulent material is reached, the incision is deepened. The extent of the abscess cavity is noted by introducing the finger into the wound. The external border of the incision is trimmed so that the wound does not close, and a rubber drain is inserted. The parents are informed that a second surgical procedure, fistulectomy, will be necessary to identify and remove the internal fistulous tract that connects the infected crypt to the abscess.

Protrusions from anal canal

When the presenting complaint is of a protrusion from the anal canal, the possibility of hemorrhoids, prolapse, procidentia, and intussusception must be considered in the differential diagnosis.

Hemorrhoids.—Internal hemorrhoids do not occur in infants, and external thrombotic hemorrhoids are rarely seen. If they are present and do not disappear in 10 days on a program of hot sitz baths, incision and removal should be carried out.

Prolapse and Procidentia.—

In both prolapse and procidentia there is abnormal descent of rectal tissue through the anal opening. In prolapse, only the mucosal and submucosal layers are involved, and the mass presents folds of mucosa running from the center radially to the periphery. In procidentia, all the walls of the rectum are involved, and the protrusion is considerably longer. The circular muscle fibers of the rectum contract the mucosa so that the surface of the elongated mass is gathered in ring-like folds which are parallel to the anal opening. Pro-

The pediatrician can do much to avoid the tragedy of an incompetent rectal sphincter by prompt diagnosis and proper treatment

truding intussusception may simulate procidentia. These lesions may be differentiated by attempting to insert a finger between the mass and the external sphincter. In intussusception displacement will occur, and the finger will be admitted into the canal; in prolapse the finger is not admitted.

The aim of treatment is to keep the prolapsing tissue in its normal position by artificial creation of supporting fibrosis in the submucosal layer and perirectal structures. In simple prolapse this is done by submucosal injection of 5 per cent phenol in oil or other sclerosing agents once a week until the situation is controlled. The predisposing factors that cause straining and, hence, prolapse, such as constipation, diarrhea, severe cough, rectal polyps, or hard crying, must be remedied if the mucosal fixation is to be maintained. The treatment of procidentia is more complicated and should be left to a proctologist.

Rectal bleeding

Rectal bleeding occurs at some point in the course of most anorectal diseases and is frequently the sign that will cause an anxious parent to bring a baby to the doctor. In the majority of cases the blood originates from some minor transitory lesion in the anorectal area, but it may also indicate the presence of a pathologic lesion hidden somewhere in the gastrointestinal tract that constitutes a threat to life. It is for this reason that the physician must make every effort to track down the source of the bleeding. Gross[3] reports sixty-five pediatric patients who were admitted to the hospital because of rectal bleeding, and after extensive investigation, which included laparotomy in some, the source of bleeding still could not be identified in eighteen.

Valuable information is gained by eliciting definite information on the history of rectal bleeding. The amount of blood passed will indicate, first, how urgent the situation is and give some indication of the type of lesion it is or is not; for example, a polyp may bleed copiously, whereas fissure in ano produces small amounts of blood.

The color of the blood gives an indication of its source. When it is bright red, the bleeding lesion usually lies in the rectum or lower colon; when it is brown, the blood has been acted on by hydrochloric acid, and the lesion is in the stomach or in contact with gastric mucosa as in Meckel's diverticulum or duplication of bowel. However, it is possible that in large hemorrhage, the blood comes down from high in the intestine so rapidly that it is still bright red when seen at the anus. Blood on the surface of the stool indicates a lesion low in the rectum; blood mixed with the body of the stool indicates a colonic lesion. Associated signs and symptoms, such as painful bleeding, bleeding only on defecation, abdominal pain, nausea, vomiting, circulatory collapse, enlargement of the spleen or liver, and hemorrhagic tendencies in other parts of the body, aid in establishing the source and seriousness of rectal bleeding.

The most common source of rectal bleeding in children is fissure in ano. There is almost always a history of constipation. Defecation is obviously painful, and crying will continue for varying periods immediately afterward. The fissure usually bleeds during defecation, streaking the surface of the stool with small amounts of bright red blood, or it may bleed just after a bowel movement.

Benign polyps are the next most frequently occurring bleeding lesions, and these may bleed copiously and usually painlessly. If in the rectum, the polyp can be palpated on digital examination. If it is higher, sigmoidoscopy or barium enema visualization is necessary for its identification.

Intussusception produces the classical "currant jelly" stool, but intermittent abdominal pain and, possibly, the presence of a palpable abdominal mass in an obviously sick baby should aid in recognition of this condition. An alarming amount of blood sometimes passes from the anus of a newborn infant within the first 48 hours after birth. This frequently is maternal blood that has been swallowed in the course of delivery and can be differentiated from fetal blood by a qualitative test described by Apt and Downey.[4]

Hemorrhagic disease of the newborn in which there is transitory hypoprothrombinemia should also be considered in this age group, and there are, of course, many other lesions which cause bleeding from the gastrointestinal tract, but the scope of this paper does not permit their discussion.

Any infant who has had one bout of bleeding which is not caused by some obvious lesion in the anus which can easily be controlled should have its blood typed and cross-matched in the event that another hemorrhage might be exsanguinating. A work-up should include proctoscopy, sigmoidoscopy, and x-ray studies of the colon and upper gastrointestinal tract. Evidence of systemic diseases with a bleeding diathesis should be sought. If the hemorrhage has been massive, or if repeated bouts of bleeding have occurred, and the lesion has not been found in the course of the work-up, the physician must then consider investigative laparotomy. However, Gross[3] performed laparotomy on four children out of eighteen in whom no cause of bleeding could be found, and in none of the four did operation reveal the cause of bleeding.

When a newborn fails to pass a meconium stool within the first 24 hours of life or there is a history of severe constipation or the passage of ribbonlike stools in an older baby, efforts should be directed toward a search for congenital anomalies of the anus and rectum. These lesions are partially or completely obstructive and may or may not have associated fistulas with the surrounding structures.

The passage of meconium stool during or shortly after the birth process is a phenomenon so taken for granted that the physician is apt to overlook its absence. Its presence must routinely be noted and the physician be alerted if it is not passed within the first 12 hours of life. At 18 hours the situation demands mobilization of an examining team, and if meconium has not yet been passed at 24 hours, a comprehensive work-up should be well under way with the diagnosis of meconium ileus or congenital bowel obstruction being strongly considered.

Anal and rectal anomalies

Various reports place the incidence of anorectal anomalies between one in 3,000 to one in 5,000. The incidence of associated anomalies such as congenital heart disease, atresia of the intestine, and esophageal atresia was 39 per cent in a series of 507 cases of anorectal malformations presented by Gross.[3] Potts[5] stresses the importance of associated genitourinary malformations and includes intravenous pyelography in his routine work-up.

Gross has classified anorectal malformations into four groups, irrespective of any associated fistulas:

Type 1 includes those patients having a patent anus or rectum but with stenosis either at the anus or in the rectum. They are brought for medical care at an average age of 9 months, the chief complaint being obstipation unless enemas or cathartics are used. The stool is ribbonlike, and the mother may complain that she has difficulty in inserting the enema tip into the baby's rectum. Examination may reveal an anal opening which is 2 or 3 mm. in diameter or an anus which appears normal, but when digital examination is made, the finger meets an annular or crescentic stricture a few centimeters above the anal orifice. The entire colon occasionally exhibits marked enlargement, and a fecal mass can be palpated through the abdominal wall.

Repeated daily dilations of the stricture will usually overcome the obstruction and permit normal function. These are performed with gum-elastic or metal dilators, the sizes of which are increased until the little finger can be introduced. When the stricture is markedly narrowed, the obstruction may have to be relieved by incision of the remaining membrane followed by dilation.

Type 2 includes those patients who have an imperforate anus, the obstruction being membranous in character. This is a complete anal block with failure to pass meconium, and symptoms of acute intestinal obstruction develop within 48 hours. Examination reveals an imperforate anal membrane which is thin enough to show darkening due to meconium lying against its inner side. The impaction of the meconium against the membrane will cause it to bulge when the baby strains or cries.

The membrane should be divided by a cruciate incision followed by occasional dilations. It is sometimes necessary to attach the mucous membrane to the skin edges with interrupted sutures.

In type 3 are those anomalies in which the anus is imperforate but the rectal pouch ends blindly some distance above it. The fistulas associated with anorectal anomalies are almost exclusively confined to this classification. In Gross's[3] series, 80 per cent of this group had rectovesical, rectourethral, rectoperineal, or rectovaginal fistulas. In the cases without fistula or when the fistula is so small that it is ineffectual in emptying the rectum, obstruction occurs and the patients resemble those with type 2 malformations. When the fistula is large enough to permit passage of feces, life is maintained; these patients were an average of 1 year of age when first seen by Gross. The complaints were of pain on defecation, constipation, or an obvious displacement of the rectal outlet.

Examination reveals that the perineum presents a small ridge or a dimple in the region where the anus should be. This depression varies in size, sometimes admitting a fingertip. The presence of a sphincter is shown by puckering of the surrounding skin when it is gently pricked with a pin. If the blind rectal pouch is close to the perineum, an impulse will be felt by a finger placed on the anal region when the baby cries. Careful probing near the vaginal fourchet may reveal a rectovaginal fistula, and the direction of the tract may give additional information concerning the position of the rectal pouch. The passage of feces or flatus through the urethra indicates a rectourethral fistula and microscopic evidence of fecal debris in a catheterized urine specimen indicates the presence of a rectovesical fistula. Rectoperineal fistulas are quite obvious on inspection of the perineum. Rectourethral and rectovesical fistulas are not found in females.

Treatment, of course, is surgical and consists of a one-stage abdominoperineal procedure in which the fistulas are tied off and the rectum is brought down through the sphincter and tacked to the skin.

In type 4 the anus and anal pouch are present and are normal; however, there is no communication between the anal and rectal canals, and the rectal pouch ends blindly in the hollow of the sacrum. Because the anus is present and appears normal, the reason for the picture of lower bowel obstruction is not as obvious as it is in types 2 and 3, and there may be considerable delay in recognizing that blockage exists within the anus. For this reason, these patients are not brought for treatment until as late as the fifth day of life and therefore constitute a much poorer surgical risk. However, digital examination will reveal a complete obstruction within easy reach of the little finger.

An abdominoperineal procedure is utilized to mobilize the entire rectum, the affected portion is discarded, and the upper rectum or lower sigmoid is joined to the anal structures.

To aid in establishing the level of complete obstruction in types 2, 3, and 4, air contrast x-ray studies should be performed routinely as described by Wangensteen and Rice.[6] A lead marker is taped on the area where the anus should be, the baby is inverted, and posteroanterior and lateral x-rays are taken. With the baby in the head-down position, the gas in the gut tract will rise to the rectal pouch and outline its distal end. Air shadows that are present distal to the blind rectal pouch are from air within the bladder and indicate the presence of a rectovesical fistula. If the films are taken within the first 24 hours after birth, the obstruction might be interpreted as existing at a level higher in the intestinal tract, when in reality the gas has not yet found its way through the sticky meconium to the end of the intestine. By the second day of life the position of the blind rectal pouch can be accurately demonstrated.

The results of therapy in Gross's[3] experience are uniformly good in types 1 and 2. In types 3 and 4 primary or secondary abdominoperineal operation resulted in a very satisfactorily functioning anus in 92 per cent of his cases. The operative mortality was 2.9 per cent; the over-all mortality was 8.6, the other deaths being attributable to serious anomalies in other parts of the body.

Conclusion

An incompetent rectal sphincter, whether due to an anomaly or to destruction by disease early in childhood, places overwhelming obstacles in the pathway to normal social adjustment. The pediatrician can, in many cases, do much to avert this tragedy by his prompt diagnosis and proper treatment of the condition.

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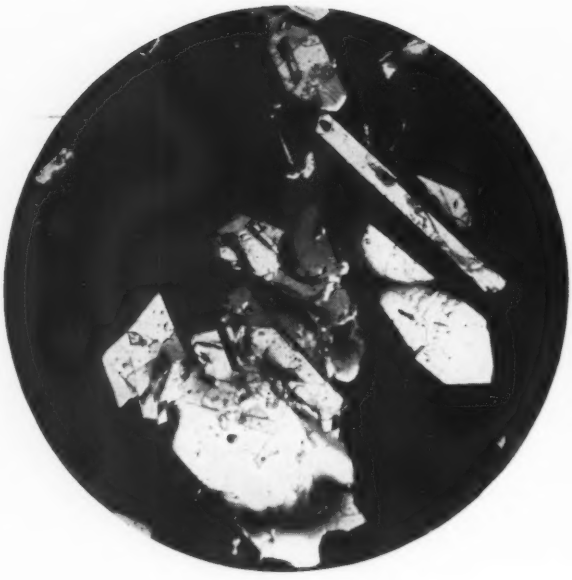

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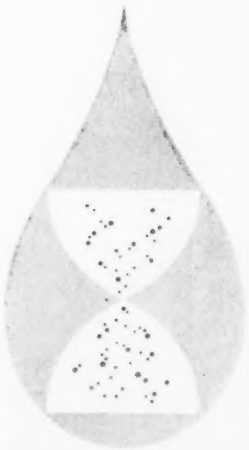

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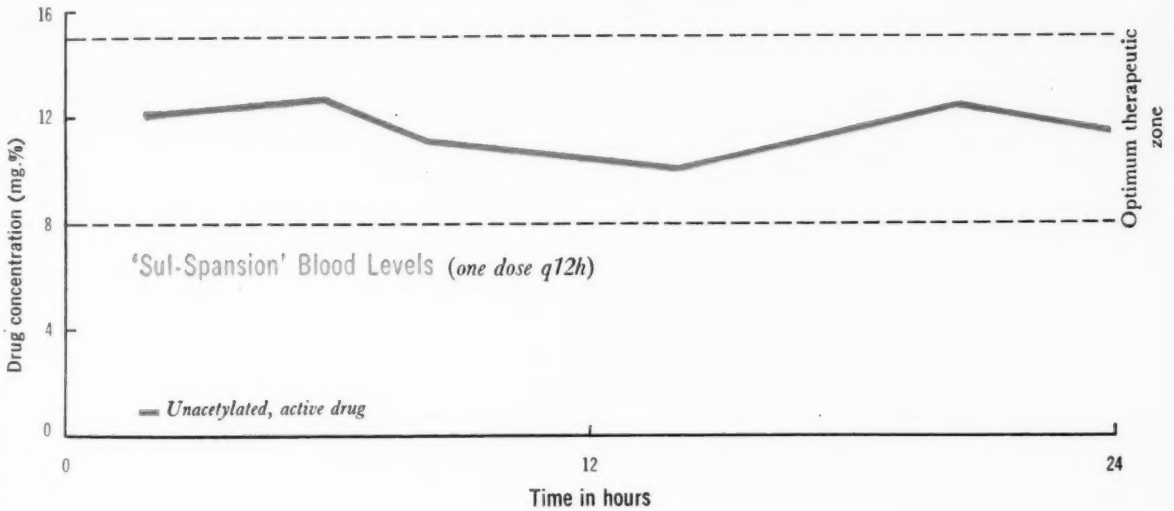

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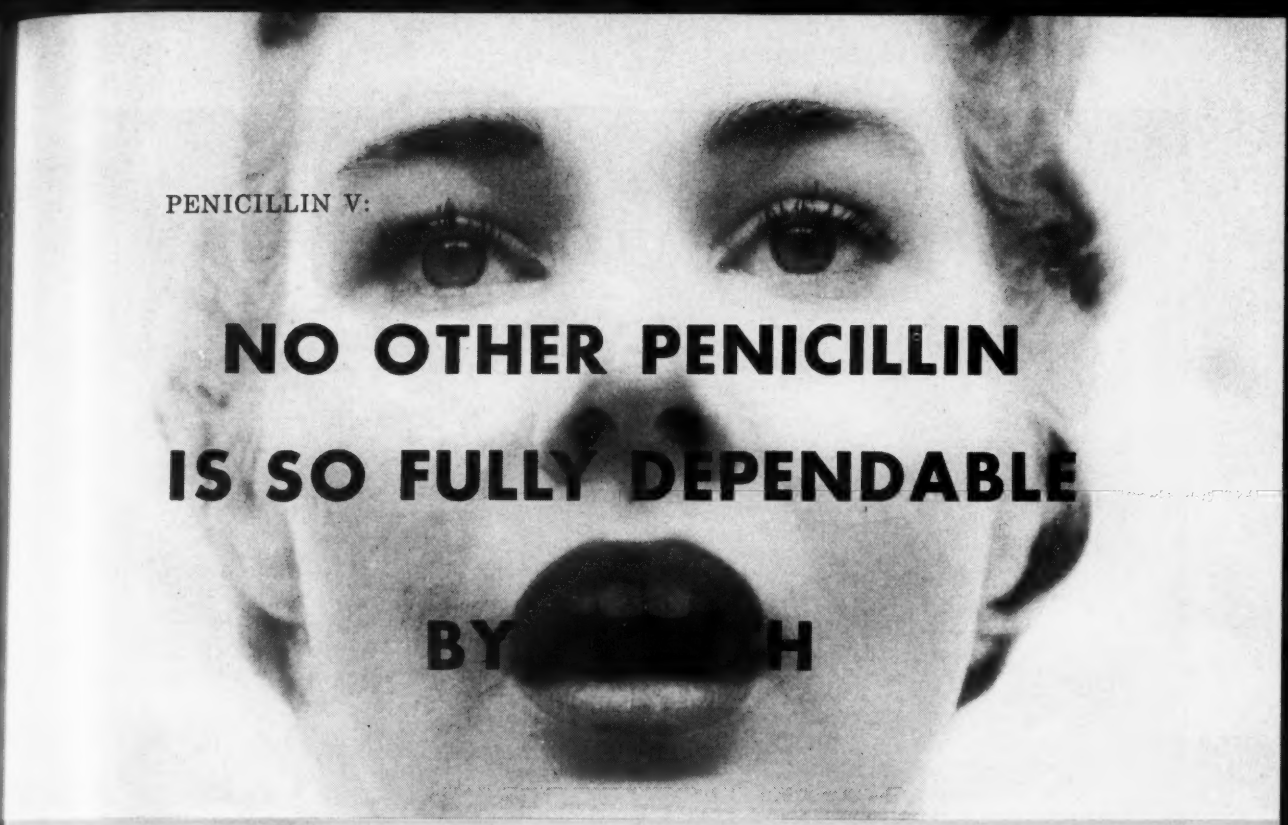

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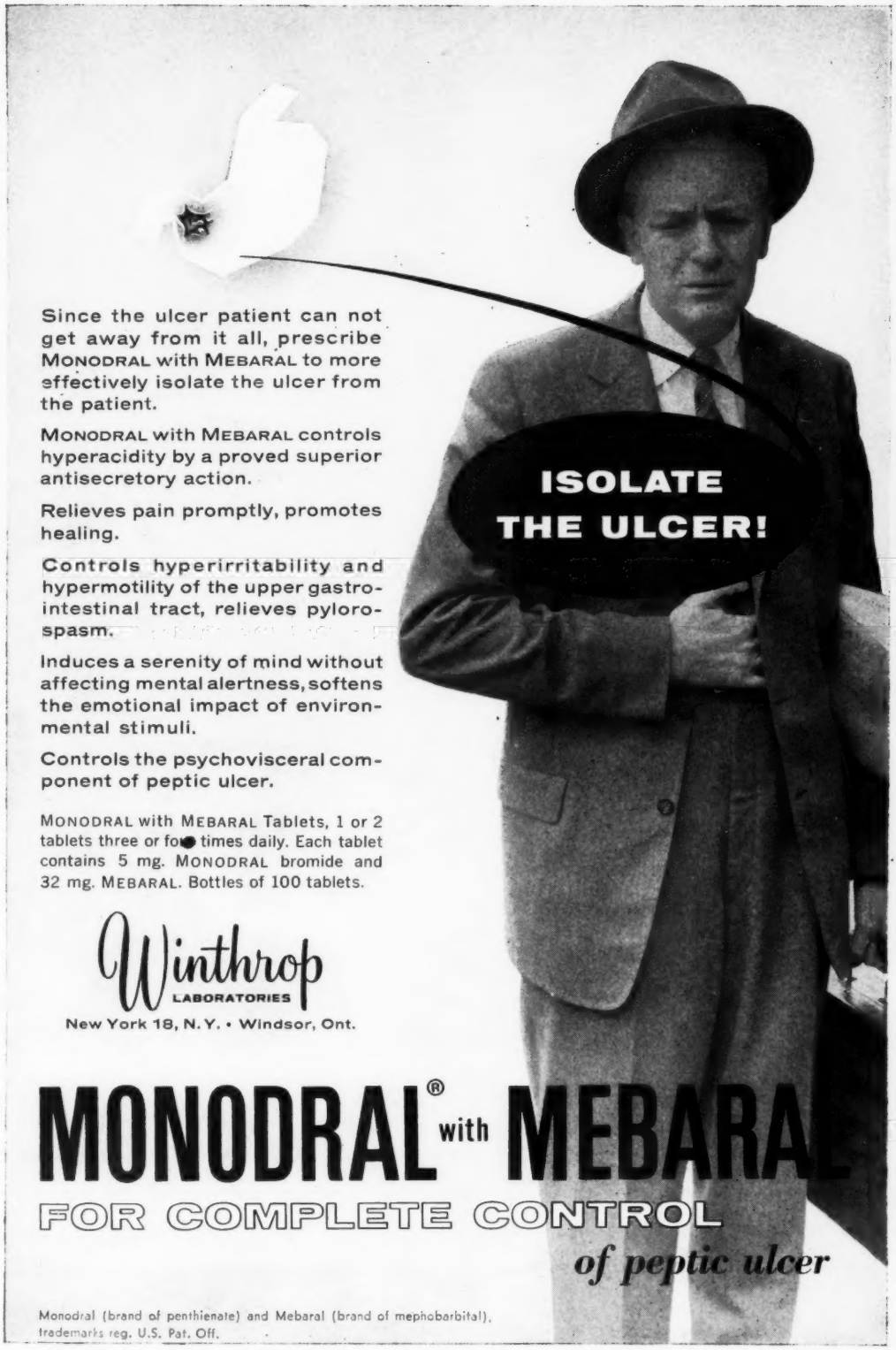

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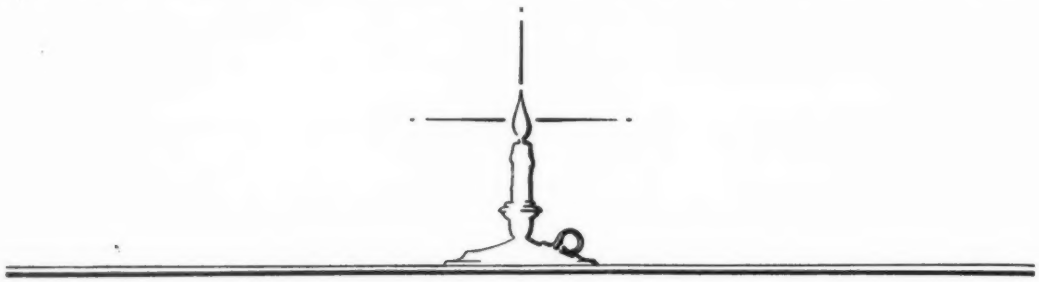

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◆ ◆ ◆

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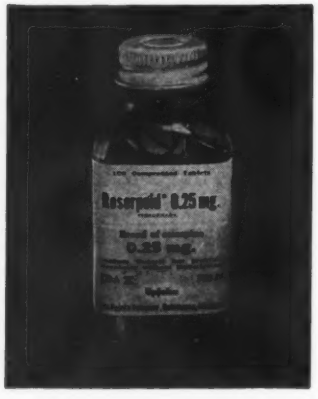

Conventions and meetings

Announcements

American Osteopathic Association, Sixty-First Annual Convention, Hotels Adolphus, Baker, and Statler, Dallas, Texas, July 15-19. Program Chairman, Neil R. Kitchen, 18820 Woodward Ave., Detroit.

American College of Osteopathic Obstetricians and Gynecologists, annual meeting, Bellevue-Stratford Hotel, Philadelphia, February 18-20. Program Chairman, P. J. MacGregor, Jr., 706 J. M. S. Bldg., South Bend 1, Ind. Secretary, Harold K. Morgan, Lakewood Polyclinic, 7575 W. 14th Ave., Denver 15.

American Osteopathic Society of Proctology, annual meeting, Hotel Statler, Detroit, April 3-5. Program Chairman, John A. Brandon, 1814 Oberlin Ave., Lorain, Ohio. Secretary, Carl S. Stillman, Jr., 3523 Fifth Ave., San Diego 3, Calif.

Arizona, annual meeting, Wigwam Hotel, Litchfield Park, April 25-28. Program Chairman, R. D. Bennett, 115 W. First St., Mesa. Secretary, Homer E. Allhouse, 2243 N. 12th St., Phoenix 22.

California, annual meeting, Hotel del Coronado, Coronado, May 15-19. Executive Secretary, Mr. Thomas C. Schumacher, 1298 Wilshire Blvd., Los Angeles 17.

Eastern Osteopathic Association, annual meeting, Hotel Statler, New York City, March 23-24. Program Chairman, Chester D. Losee, 212 Prospect St., Westfield, N.J. Secretary, Frank B. Tompkins, 309 Baltimore Life Bldg., Baltimore 1, Md.

Florida, annual meeting, Jacksonville, September 30-October 2. Program Chairman, Louis J. Larmoyeux, 124 E. Ashley St., Jacksonville 2. Secretary, Dominic Raffa, 5009 Central Ave., Tampa 3.

Georgia, annual meeting, King and Prince Hotel, St. Simons, May 9-11. Program Chairman, Walter B. Elliott, Jr., 702 Mortgage Guarantee Bldg., Atlanta 3. Secretary, Ruth McNeff Glass, 1432 Donnelly Ave., S.W., Atlanta 10.

Illinois, annual meeting, Palmer House, Chicago, April 26-28. Program Chairman, S. V. Robuck, 25 E. Washington St., Chicago 2. Executive Secretary, Mr. D. O. Durkin, Rm. 521, 53 W. Jackson Blvd., Chicago 4.

Indiana, annual meeting, Marott Hotel, Indianapolis, May 19-21. Program Cochairmen, Paul van B. Allen, 1500 N. Delaware St., Indianapolis 2, and H. E. Rinne, 519-20 Merchants Bank Bldg., Indianapolis 4. Secretary, Arabelle B. Wolf, 4840 N. Michigan Rd., Indianapolis 8.

Iowa, annual meeting, Savory Hotel, Des Moines, May 27-28. Program Chairman, R. S. Farran, Badgerow Bldg., Sioux City 9. Secretary, Mr. Herman W. Walter, 200 Walnut Bldg., Des Moines 9.

Kansas, annual meeting, Town House, Kansas City, May 4-8. Program Chairman, V. R. Cade, Cade Clinic Bldg., 816-818 Broadway, Larned. Secretary, Mr. Lloyd L. Hall, 121 E. 8th St., Topeka.

Maine, annual meeting, Samoset Hotel, Rockland, June 27-29. Professional Education Chairman, Donald P. Miller, Main St., Norridgewock. Secretary, Roswell P. Bates, 72 Main St., Orono.

Massachusetts, annual meeting, Somerset Hotel, Boston, January 19-20. Program Chairman, William A. Seeglitz, 315 Franklin St., Newton 58. Secretary, Mrs. Gladys M. Stockdale, 524 California St., Newtonville 60.

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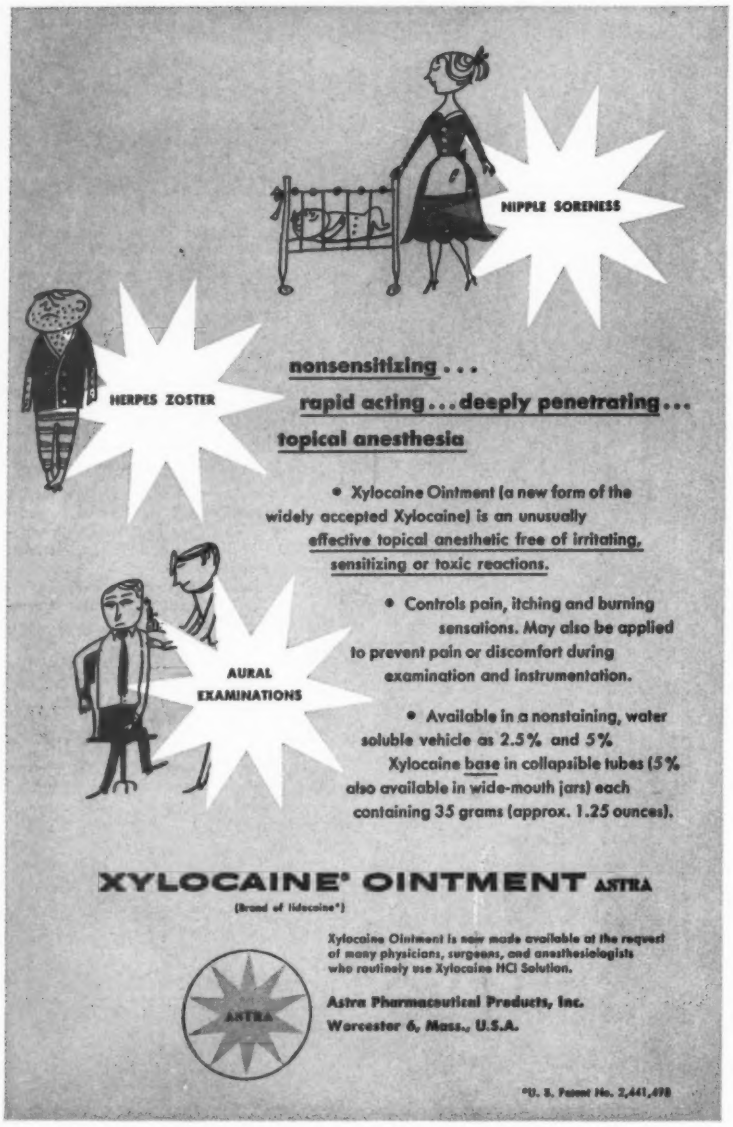
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*U. S. Patent No. 2,441,498

Michigan, annual meeting, Grand Rapids, September 30-October 2. Program Chairman, Harold W. Laidlaw, McCoy-Laidlaw Clinic, Bad Axe. Secretary, P. Ralph Morehouse, 214 S. Superior St., Albion.

Minnesota, annual meeting, Lowry Hotel, St. Paul, May 2-4. Program Chairman, M. Sidney Hedeen, 1595 Selby Ave., St. Paul 4. Secretary, E. R. Komarek, 301 Granite Exchange Bldg., St. Cloud.

New Jersey, annual meeting, Ritz-Carlton Hotel, Atlantic City, March 2-3. Program Chairman, Kirk L. Hilliard, 850 N. Main St., Pleasantville. Secretary, Richard F. Leedy, 38 Newton Ave., Woodbury.

New Mexico, annual meeting, Navajo Lodge, Ruidoso, May 16-18. Program Chairman, Lory Baker, 400 N. Church St., Las Cruces. Secretary, Robert E. Smith, 205 N. First, Lovington.

Northwest Osteopathic Convention, Multnomah Hotel, Portland, Oregon, June 17-20. Program Chairman, Russell F. Kenaga, 2265 W. Burnside St., Portland 5, Ore.

Ohio, annual meeting, Neil House Hotel, Columbus, May 5-8. Program Chairman, Donald Siehl, 701 Salem Ave., Dayton 6. Secretary, Mr. William S Konold, 53 W. Third Ave., Columbus.

Ontario, annual meeting, Orillia, May 2-4. Program Chairman, C. V. Hinsperger, 806 Canada Bldg., Windsor. Secretary, A. V. DeJardine, 205 Yonge St., Toronto 1.

Oregon: See Northwest Osteopathic Convention.

South Dakota, annual meeting, Huron, June 2-3. Program Chairman, F. A. Nutter, Jr., Box 368, Beresford. Secretary, Earl W. Hewlett, 417 W. 27th St., Sioux Falls.

Tennessee, annual meeting, Graystone Hotel, Gatlinburg, April 28-May 1. Program Chairman, L. D. Elliston, 707 S. Main St., Covington. Secretary, J. M. Moore, Jr., 200 High St., Trenton.

Texas, annual meeting, Plaza Hotel, San Antonio, May 2-4. Program Chairman, Myron S. Magen, 5101 Ross Ave., Dallas 6. Secretary, Phil R. Russell, 512 Bailey St., Fort Worth 7.

Washington: See Northwest Osteopathic Convention.

West Virginia, annual meeting, Daniel Boone Hotel, Charleston, June 2-4. Program Chairman, Frank E. Pick, 4410 McCorkle Ave., S.W., South Charleston 3. Secretary, Guy E. Morris, 542 Empire Bank Bldg., Clarksburg.

Wisconsin, annual meeting, Wisconsin Hotel, Milwaukee, April 29-May 1. Program Chairman, I. J. Ansfield, 1500 E. Brady St., Milwaukee 2. Secretary, Edwin J. Elton, 1518 N. 70th St., Wauwatosa 13.

State and national boards

ARIZONA

Those interested in professional examinations should contact Russell Peterson, D.O., secretary, Osteopathic Board of Registration and Examination in Medicine and Surgery, 2747 East McDowell Road, Phoenix.

Basic science examinations March 19 at the University of Arizona, Tucson. Applications must be filed 2 weeks prior to examination. Address H. D. Rhodes, Ph.D., secretary, Basic Science Board, University of Arizona, Tucson.

COLORADO

Basic science examinations in March. Address Esther B. Starks, D.O., secretary, Basic Science Board, 1459 Ogden St., Denver 18.

CONNECTICUT

Basic science examinations February 9. Address State Board of Healing Arts, 52 Whitney Ave., New Haven 10.

Professional examinations March 12. Address Frank Poglitsch, D.O., secretary, Osteopathic Examining Board, 300 Main St., New Britain.

DELAWARE

Examinations January 8-10. Address Joseph S. McDaniel, M.D., secretary, Board of Medical Examiners, 229 S. State St., Dover.

HAWAII

Examination dates by Territorial Law are usually the first Wednesday, Thursday, and Friday of January, April, July, and October. One of these dates is set for examination after approval of candidate's application by the Board. Address Frank O. Gladding, D.O., secretary, Board of Osteopathic Examiners, 504 Hawaiian Trust Bldg., Hawaii 13.

ILLINOIS

Examinations in January in Chicago. Address Mr. Frederic B. Selcke, Superintendent of Registration, Department of Registration and Education, State House, Springfield.

IOWA

Basic science examinations January 8 at the Capitol Bldg., Des Moines. Address Ben H. Peterson, Ph.D., secretary, Board of Basic Science Examiners, Coe College, Cedar Rapids.

KANSAS

Examinations February 14-15 at the Jayhawk Hotel, Topeka. Address C. A. Welker, D.O., secretary, Board of Osteopathic Examination and Registration, J. C. Penny Bldg., Concordia.

MASSACHUSETTS

Examinations January 15-18 at State House, Boston. Applications must be filed by January 2. Address Robert C. Cochrane, M.D., secretary, Board of Registration in Medicine, Room 37, State House, Boston 33.

MICHIGAN

Basic science examinations February 8 and 9. Applications must be filed by January 31. Address Mrs. Anne Baker, secretary, Board of Examiners in the Basic Sciences, 116 Mason Building, Lansing.

MONTANA

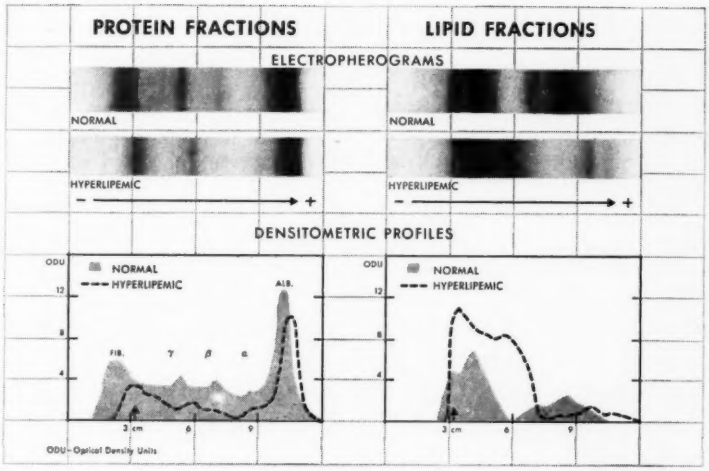
Examinations March 5. Address Asa Willard, D.O., secretary, Board of Osteopathic Examiners, Wilma Bldg., Missoula.

NEBRASKA

Basic science examinations January 8-9. Address Mr. Husted K. Watson, Director, Bureau of Examining Boards, Department of Health, State Capitol Bldg., Lincoln 9.

NEVADA

Professional examinations January 26. Applications must be filed by January 12. Address Walter J. Walker, D.O., secretary, Board of Osteopathic Examiners, 210 W. Second St., Reno.



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Basic science examinations January 8. Address Donald G. Cooney, Ph.D., secretary, Board of Examiners in the Basic Sciences, Box 9005, University Station, Reno.

NEW YORK

Examinations February 5-8, in Albany, Buffalo, New York City and Syracuse. Applications must be filed January 7. Address John W. Paige, M.D., Chief, Bureau of Professional Examinations & Registrations, 23 S. Pearl St., Albany 7.

NEW MEXICO

Basic science examinations January 20. Address Mrs. Marguerite Cantrell, secretary, Board of Examiners in the Basic Sciences, P.O. Box 1522, Santa Fe.

NORTH DAKOTA

Examinations in January. Address G. L. Hamilton, D.O., secretary, Board of Osteopathic Examiners, Ringo Bldg., 119 S. Main St., Minot.

OREGON

Professional examinations in January. Address Mr. Howard I. Bobbitt, 609 Failing Building, Portland.

RHODE ISLAND

Professional examinations January 3-4. Address Mr. Thomas B. Casey, Administrator of Professional Regulation, 366 State Office Bldg., Providence.

Basic science examinations February 13. Applications must be filed 21 days in advance. Address Mr. Casey.

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SOUTH DAKOTA

Professional examinations January 15-16 at Sioux Falls. Address Mr. John C. Foster, executive secretary, Board of Medical and Osteopathic Examiners, Room 300, First National Bank Bldg., Sioux Falls.

TENNESSEE

Examinations in February and July at Nashville. Applications must be filed 15 days prior to examination. Address M. E. McCoy, D.O., secretary, Board of Examination and Registration for Osteopathic Physicians, 1226 Highland, Jackson.

VERMONT

Examinations January 23-24, State House, Montpelier. Applications must be filed by January 10. Address Charles D. Beale, D.O., secretary, Board of Osteopathic Examination and Registration, Mead Bldg., Rutland.

WASHINGTON

Professional examinations January 14-16, University of Washington, Seattle. Applications must be filed 15 days prior to examination. Address Professional Division, State of Washington Department of Licenses, Olympia.

Basic science examinations January 9-10, University of Washington, Seattle. Applications must be filed 15 days prior to examination date. Address Professional Division, Washington State Department of Licenses, Olympia.

WISCONSIN

Examinations January 8 at Madison. Address Thomas W. Tormey, Jr., M.D., secretary, Board of Medical Examiners, State Office Bldg., 1 West Wilson St., Madison.

WYOMING

Examinations February 4. Address Franklin D. Yoder, M.D., New State Office Bldg., Cheyenne.

Reregistration of osteopathic licenses

December 31—Tennessee, $5.00. Address M. E. Coy, D.O., secretary, Board of Examination and Registration for Osteopathic Physicians, 1226 Highland, Jackson.

Prior to January 1—Arizona, not more than $10.00. Address Russell Peterson, D.O., secretary, Osteopathic Board of Registration and Examination in Medicine and Surgery, 2747 E. McDowell Rd., Phoenix 22.

January 1—California, $20.00 for residents and nonresidents. Address Glen D. Cayler, D.O., secretary, Board of Osteopathic Examiners, 1013 Forum Bldg., Sacramento 14.

January 1—Florida, $5.00. Address W. S. Horn, D.O., secretary, Board of Osteopathic Medical Examiners, 1500 Eighth Ave., Palmetto.

January 1—Maine, $4.00. Address George F. Noel, D.O., secretary, Board of Osteopathic Examination and Registration, Monument Square, Dover-Foxcroft.

January 1—Manitoba, $5.00. Address W. Kurth, D.O., secretary, Board of Osteopathic Physicians, 248 Moorgate Blvd., Deer Lodge, Winnipeg.

January 1—New York, $6.00, biennially. A physician receiving a license the second year of any biennial registration period pays a fee of $3.00 for a certificate expiring December 31 of such second year. Address Stiles D. Ezell, M.D., secretary, Bureau of Professional Examinations and Registrations, 23 S. Pearl St., Albany 7.

January 1—Ontario, $35.00. Address D. Gordon Campbell, D.O., secretary, Board of Directors of Osteopathy, 2 Bloor St., E., Toronto 5.

January 1—Pennsylvania, Surgeons Examining Board, $10.00. Address Mrs. Katherine M. Wollet, acting secretary, Board of Osteopathic Examiners, Bureau of Professional Licensing, Harrisburg.

January 1—Texas, $5.00. Address M. H. Crabb, M.D., secretary, Board of Medical Examiners, 1714 Medical Arts Bldg., Forth Worth 2.

January 1—Utah, $3.00. Address Alice E. Houghton, D.O., secretary, Osteo-

pathic Examining Board, 600 Zion's Saving Banks Bldg., Salt Lake City 1.

January—Alberta. No reregistration. Pay $10.00 a year membership in the College of Physicians and Surgeons, Alberta, in January.

During January — Connecticut, $2.00. Address Frank Poglitsch, D.O., secretary, Osteopathic Examining Board, 300 Main St., New Britain.

During January—Minnesota, $2.00. Address Wallace F. Kreighbaum, D.O., secretary, Board of Osteopathic Examiners, 2748 Hennepin Ave., Minneapolis 2.

During January—Wisconsin, $3.00. Address Thomas W. Tormey, Jr., M.D., secretary, Board of Medical Examiners, State Office Bldg., 1 W. Wilson St., Madison.

January 31—British Columbia, amount of fee set at Annual Meeting of Council of College of Physicians and Surgeons of British Columbia. Address Lynn Gunn, M.D., 1807 W. 10th Ave., Vancouver 9.

Prior to February 1—Vermont, $3.00 residents, $2.00 non-residents. Address Charles D. Beale, D.O., Meade Bldg., Rutland.

Before March 1—Colorado, $2.00 if legal resident of Colorado; $10.00 if not legal resident of Colorado. Address Mrs. Beulah H. Hudgens, executive secretary, State Board of Medical Examiners, 831 Republic Bldg., Denver 2.

Examination by National Board

The National Board of Examiners for Osteopathic Physicians and Surgeons conducts Parts I and II of its examination on the first Thursday and Friday of each May and December at the six approved colleges. Application blanks may be obtained from the secretary or the dean of the college, and the completed application blank, together with a passport photograph and check for the parts to be taken, must be in the secretary's office by the November 1 or April 1 preceding the examination.

Examinations in Part I consist of anatomy, including histology and embryology; physiology; physiological chemistry; general pathology; and bacteriology, including parasitology and immunology.

Part II consists of examinations in surgery, including applied anatomy, surgical pathology, and surgical specialties; obstetrics and gynecology; pediatrics, neurology and psychiatry; public health, including hygiene, medical jurisprudence; osteopathic principles, therapeutics, including pharmacology and materia medica.

Part III is an oral and practical examination given under the supervision of a chief examiner who is a member of the Board and by a panel of associate examiners. Subjects covered in Part III are anatomy; physiology; pathology; osteopathic principles; therapeutics, and pharmacology; surgery; ophthalmology and otorhinolaryngology; obstetrics and gynecology; physical and clinical diagnosis; public health and communicable diseases.

Part III examinations will be given at Philadelphia, March 2-3, at Kirksville, Mo., March 10, and at Los Angeles, March 16-17. Applications must have been filed with the secretary of the Board by January 1.

These are oral examinations which the candidate may take after having satisfactorily completed the first 6 months of a 1 year internship in a hospital approved by the American Osteopathic Association for intern training. Part III is given annually at the Philadelphia, Kirksville, and Los Angeles colleges.

Eligibility requirements are as follows: Part I, satisfactory completion of the first 2 years in an approved school of osteopathy; Part II, satisfactory completion of Part I and of the first two quarters or trimesters of the senior year in an approved osteopathic college; Part III, satisfactory completion of Part II and at least 6 months of a 1-year internship approved by the American Osteopathic Association. The internship requirement does not apply to candidates who took Part I prior to July, 1950.

Applications must be filed with the secretary of the Board not less than 30 days prior to the examination dates. Address Paul van B. Allen, D.O., secretary, 1512 N. Delaware Street, Indianapolis 2, Indiana.

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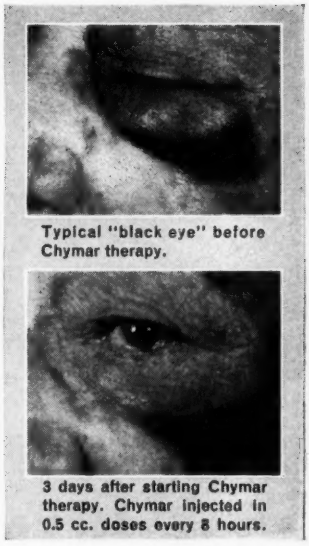

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What it does—

Chymar controls inflammation and restores normal circulation. It hastens absorption of hematomas, minimizes tissue necrosis and promotes healing.

Why Chymar is so safe—

There are no systemic side effects with Chymar. Chymar does not interfere with blood clotting, and no clotting time or serum protein determinations are necessary. There are no known contraindications to Chymar and no known incompatibilities.

Indications: Chronic ulcers (stasis, varicose, diabetic); reduction of hematomas; swelling due to trauma; cellulitis; bursitis and arthritis; phlebitis; and inflammation of the eye (iritis, iridocyclitis, chorioretinitis, uveitis).

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America's children*

Within the single decade following World War II, the child population of the United States increased by more than one third, reaching a record total of 55⅔ million at ages under 18 years in 1955. This rise is without precedent in our history, the gain in the 10 years amounting to more than 14 million. The situation reflects essentially a new outlook toward family life by our young people—the proportion married is at an all-time high, and they are marrying and starting their families earlier.

The growth of our child population during this century is marked. From 1900 to 1930, the number of children under 18 years of age rose to 43 million. The increase during these three decades, amounting to nearly 12⅓ million, was actually less than that in the past decade. In contrast, the number of children declined after 1930 and was only 41½ million in 1945. This drop shows the effect upon our population structure of the low birth rates during most of the intervening period.

Examination of the detail shows that the postwar upsurge in the number of children is concentrated very largely at ages under 10 years. Thus, from 1945 to 1955, the number at ages under 5 years rose by 41 percent and at ages 5-9 years by 58 percent. The increase at ages 10-14 was only 24 percent, while at ages 15-17 years there was actually a small decrease. The latter are the children who were born just before World War II, when the birth rate was still relatively low. Within the next few years, however, the number of teen-agers will rise rapidly as the large cohort of younger children grows older.

Most of our children have the benefits of a normal family life. According to available data, in 1955 almost 48 million children under 18 years of age—about 7 out of every 8 of that age—were living with both parents. An additional 600,000 were living with only the father present in the household, and almost 5.2 million with the mother only. The latter include about one-half million children whose fathers were absent because of service in the Armed Forces. Somewhat under one million children were living with relatives other than parents, and less than one-half million under other arrangements.

The high proportion of children living with both parents reflects, in part, the favorable mortality trend in the country. This has saved a great many children from orphanhood and kept for them the advantages of care and support by both parents. One generation ago, in 1920, about 6.4 million children under 18 years of age—16 percent of the total—had lost one or both parents by death. In 1955 only 2.7 million children—less than 5 percent of the total—were orphaned, and the great majority were living with the surviving parent.†

Education of children is highly valued in American life. According to a report from the Bureau of the Census, in October 1955, 78 percent of the children at ages 5 and 6 were already attending school. This proportion rose to more than 99 percent at ages 7-13 years, fell somewhat to 96 percent at ages 14-15, and dropped further to 77½ percent at ages 16-17 years.

The decrease in the proportion attending school among late teen-age children reflects their entry into economic life with the approach of maturity and, for girls, marriage and motherhood. In addition to those who leave school, a great many in their teens work part time outside of school hours or during vacation periods. However, less than one fourth of all students of ages 14-17 years are in the labor force while still in school. The average number of employed teen-agers in and out of school was about 2¼ million during 1955. They represented one sixth of all children at ages 14-15 years, and one third at ages 16-17 years. These figures include a small number of boys aged 17 years serving in the Armed Forces. Among the girls, about 10 percent at ages 16 and 17 years are married. Each year, about 140,000 girls under 18 years of age become mothers, some bearing their second or third child.

*Reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, August 1956.

†*Social Security Bulletin*, March 1955, p. 18, and February 1956, p. 11.

The physician and the family*

The character of the professional relationships of a physician both with the families of his patients and with his associates in health education and family care will be affected directly by the kind of education he receives in medical school. Recently developed teaching projects designed to advance the practice of health education in family living are evidence of a trend toward educating the physician to understand the health of the patient in relation to the family environment.

The origins and reasons for this movement are complex; their contributors, both individual and organizational, are many. In the past quarter of a century, the Association of American Medical Colleges particularly has emphasized the importance of appraisal of social and environmental factors in clinical teaching. The 5-day teaching institute held in 1952 by the Conference of Professors of Preventive Medicine and the Association of American Medical Colleges at Colorado Springs, Colo., gave considerable impetus to the further expansion of comprehensive medical care teaching demonstrations, with emphasis on home and ambulant care.

Of interest is the extent to which departments of preventive medicine have a responsible role in this form of extramural teaching, the appearance of psychiatry in a consultative teaching function on behalf of other departments, and new administrative arrangements and purposes in the relations of clinical departments for the demonstration of medical care. The Cornell comprehensive care and teaching program and Boston University's domiciliary medical care program are good examples of the latter.

THE STATE UNIVERSITY PROJECT

As a learning device and as a form of service, the value of simultaneous appraisal of all members of a household has been demonstrated in the Family Health Study Program of the State University of New York College of Medicine at New York City. This family medical and social appraisal is the major project of a 1-month full-time clerkship in environmental medicine and community health carried on at the health department's Red Hook-Gowanus District Health Center in Brooklyn, N. Y. Each senior medical student is assigned a family chosen for the program by the health center's community nursing service and as much of the study as possible is conducted within the family home in a series of frequent visits.

Physicians, social workers, public health nurses, social scientists, and staff of community agencies serve as consultants to the student and evaluate his findings with him individually and in groups. The recommendations found most acceptable in the joint conferences are acted upon by the public health nurse in the further use of community agencies for continuing care.

The totality, size, and concentrated time span of the family studies of this program differ notably from what is usual in family medical practice where the physician's contact with a family is usually episodic and in response to a call for the care of the family member who is ill. In the State university study program all family members are equally the object of attention in the same time period. Some of the examinations performed go beyond those commonly made by a practicing physician; for example, after the student-family relationship has become stabilized, examination may be made of the home to discover hazards that might cause accidents.

THE STUDENT'S APPRAISAL

In making his report, the student appraises each member of the family at three levels, according to the individual's personal health, the latter's role in and relations with other members of the family, and his role in and relations with the community itself. The need of reducing the record to diagnostic and summary terms leads into the consideration of several general questions of instructional importance. One family's history illustrates two such questions: *(a)* the problem of selecting the initial steps and the

*Brief of paper presented at 16th Eastern States Health Education Conference, New York City, April 1956, by Duncan W. Clark, M.D., professor and chairman, department of environmental medicine and community health, College of Medicine, State University of New York, N.Y. Reprinted from *Public Health Reports*, October 1956.

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frequently limited goals possible in aiding the restoration or promotion of family functioning and personal health and *(b)* the opportunity of examining the family for identification of diseases which have a familial risk.

The husband, an unskilled laborer, had been unemployed for a year. He had asthma and bronchitis, and official agencies were about to investigate the possibility that these conditions were sufficiently disabling to justify the transfer of financial support of the family from the category of home relief (for the fully employable) to that of aid to dependent children. He was found to be depressed, showed striking symptoms of self-devaluation, and his relations with his wife had deteriorated to the point of consideration of divorce.

Although the wife had not handled the family funds when the husband was the provider, she had since taken over the handling of public assistance funds. She seemed competent as a mother, but her ability as a wife was more open to question. Perhaps childhood illness and experiences, or the lack of much of a childhood—she had worked in a factory from age 8 to 18 years—may have qualified her ability to relate on a mature level.

A problem such as the unemployment of the head of the family offers a series of choices in the attempt at solution. Determined agency activity in finding the husband a job might restore his status again as the main provider and head of the household. Referral of the wife to a family agency for a fuller understanding of her situation and of her attitudes toward her husband and even of her version of his behavior might identify which adult member in the family is the better able to form a relationship. Or the husband might be referred to a psychiatrist for an examination to determine whether his behavior is evidence of a psychological disorder that may disqualify him for work or for certain kinds of work.

The nature of the training and experience of health personnel may be the main determinants of the choice of one of these directions toward more healthful functioning. The physician, the social worker, and the public health nurse each select a different approach to this family's problem. Further, there are times when vocational advice may require inquiry into the health status of family members and understanding of family relationships.

FAMILY DISEASE AND FAMILIAL RISK

In the case of an individual or family a doctor's concern must include attention to resistance and susceptibility to disease. Ideally, in a full appraisal and in anticipating susceptibility to disease, inquiry needs to be extended to all relatives, living and dead, to pregnancies unfulfilled, and even to the yet unborn. Such a case study approach will provide students and teachers with data for a discussion of existing knowledge, or lack of knowledge, of the mechanisms of disease as influenced by genetics and by environment.

As a teaching device we may conceive "family disease" as multiple cases of a disease among relatives or single cases of disorders known to carry a "familial risk" to children. In the family mentioned previously there were two likely instances of disorders associated with familial risk to children. The mother had dextrocardia and 1 of the 5 children had epilepsy. Two maternal aunts and the maternal grandmother had gallstones, but it is not known whether the mother had gallstones as well. She had eclampsia on one occasion and her mother had hypertension. Little of significance can usually be drawn from the history of more distant relatives. On the subject of disorders known to be common, there were nutritional deficiencies, and these were presumed to be due to the family's marginal economic circumstances.

The term "familial risk" denotes an empirical observation without the usual assumption of genetic etiology. Families share common environments as well as common genes, and families in consecutive generations show some tendency to remain within the same cultural, religious, and economic groups. In the family studied, two possible familial risks were present, epilepsy and dextrocardia. Because the parents in this family were cousins, the question was pertinent whether children as yet unborn to this marriage might be heir to dextrocardia. However, since as many as five children had been born without this rare congenital defect there is little probability of a later child having the condition.

ATTITUDES AFFECTING COUNSELING

The possible role of personal and professional attitudes in counseling may be

illustrated in a study of a family of four. The father had advanced pulmonary tuberculosis, one child had recently recovered from chorea, and the other child had had rheumatic fever for 2 years and had been in a special institution for this disease. The mother had gone to work 6 months earlier, at the time of her husband's hospitalization. There had been no untoward consequences to the children with the mother at work, but the question was raised speculatively in conference whether she should have made this decision.

The 16 medical students unanimously agreed that the mother should have gone to work. But medical students believe in hard work, and they have a strong pragmatic streak. After all, they seemed to say, the woman had been working for 6 months with no serious consequences to herself or her children.

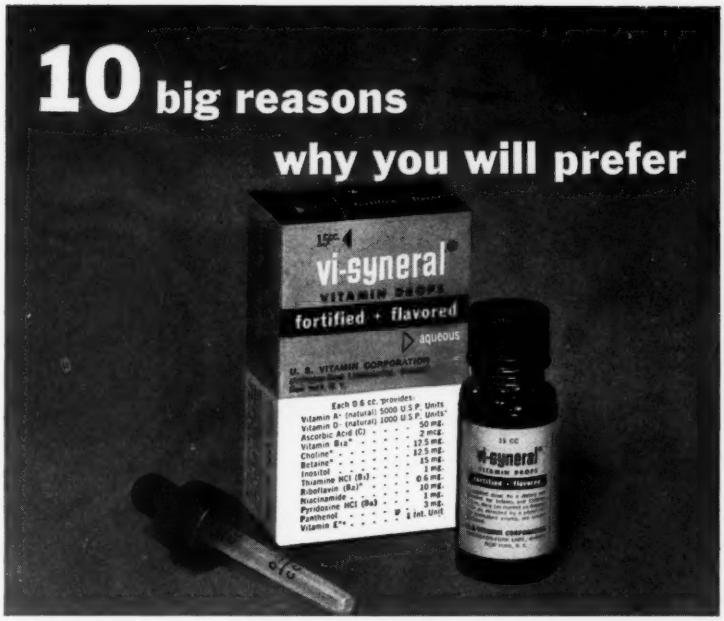
The intended implication of the question was what response representatives of special fields might make to the question of the advisability of the mother's employment, according to their professional knowledge and orientation. The following responses might have been made.

A pediatrician might be primarily concerned with the fact that two young, recently convalescent children were unattended on their return from school.

A psychiatrist might feel that the wife's pursuit of work, while relieving anxiety in her, could have an emasculating effect on the husband and could pressure him to a premature return to work.

An anthropologist might say that it is traditional for Puerto Rican women to work hard and that the mother's employment was a decision acceptable to both sexes of this ethnic group.

An internist might feel that prevention of tuberculosis in the mother is the critical issue and that her undoubted recent exposure to the disease should weigh against her working, although his opinion might be based more on clinical prudence than on possession of the facts on the role of work.



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Fatal accidents among cyclists*

Youngsters 5 to 14 years of age account for two thirds of the approximately 600 lives lost in the United States each year in accidents involving bicyclists. This death toll could be greatly reduced if cyclists obeyed the traffic rules and kept their vehicles in good running condition. Inasmuch as many of these riders are too young to be familiar with traffic regulations or aware of their significance, motorists need to exercise particular caution when they meet cyclists on the road or when they drive through residential and other areas frequented by bicyclists.

Some detail regarding bicycle accidents is available from the death claim records of 151 Industrial policyholders of the Metropolitan Life Insurance Company who were killed in this way during the four years 1952-55. Males accounted for 137 of these deaths; this was nearly 10 times the number among females. Half of the fatalities among the males were concentrated in the five-year age group 10-14; an additional one fourth occurred in the two adjacent age groups, 5-9 and 15-19, combined. Among females, all the deaths were at ages under 15 years years.

Collisions with motor vehicles were responsible for four out of every five deaths in this study. Most of these collisions were with passenger automobiles. Trucks and buses were involved in one third of the bicycle-motor vehicle collisions. It is possible that the larger vehicles may cause undue confusion among cyclists, particularly the younger ones.

A wide variety of dangerous actions by cyclists were reported as factors in bicycle-motor vehicle collisions. The most common violations of safe riding were swerving into the path of a motor vehicle, coming out of a driveway without exercising caution, entering a traffic lane from behind a parked car, and racing with other cyclists. Some deaths were attributed to cyclists disregarding stop

*Reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, August 1956.

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References: 1. Kass, E. H.: Am. J. Med. 18:764, 1955. 2. Diggs, E. S., Prevost, E. C., and Valderas, J. G.: Am. J. Obst. 71:399, 1956.

signs or other safety regulations at street intersections. A number of the bicycles struck by motor vehicles during the hours of darkness were without reflectors or lights.

In other instances it appears, from the information reported, that much of the responsibility for the fatal accident rested with the motor vehicle operator. A few deaths were attributed to hit-and-run motorists.

The most frequent types of fatal bicycle accidents, other than those arising from collision with motor vehicles, were those in which riders fell or were thrown from bicycles of which they lost control; about one eighth of the persons in this study were killed that way. The various circumstances under which these deaths occurred included carrying extra passengers; some mechanical defect, such as a broken pedal or a loose front wheel; stalling suddenly in soft ground; and striking small objects on the road. A few of the victims were drowned when they lost control of their vehicles on a river bank or a small bridge. Other types of accidents involved collisions between two bicycles or with fixed objects, such as stone walls or poles. A small proportion of the fatally injured were pedestrians struck by cyclists.

The National Safety Council, the Bicycle Institute of America, school and traffic authorities, and other groups have done much to make children aware of the hazards that attend cycling and to teach them safe riding habits. Parents can also play an important role in the prevention of bicycle accidents by stressing safe practices to their children and the need of observing them.

Water fluoridation

A positive health protection*

Frank E. Law, D.D.S., M.P.H.

Chief, Operational Research Branch, Division of Dental Public Health, Public Health Service, Department of Health, Education and Welfare, Washington, D. C.

Nearly every decade of the past century has marked a milestone in the progress of public health—a triumph over one of the hazards to the health of the people. Milk pasteurization, water chlorination, anesthesia, the antibiotics, the Salk vaccine, are only a small number of the list of successes. Water fluoridation, for the partial prevention of tooth decay, deserves a prominent place in any such listing of public health achievements.

Water fluoridation is the adjustment of the fluoride content of a water supply to a level which reduces the incidence of tooth decay. This optimum concentration is very low—one part of fluoride for each million parts of water in most areas of the country. Sodium silicofluoride, sodium fluoride, and hydrofluosilicic acid are the most commonly used fluoride compounds. These are fed into the water by equipment similar to that used for years to add chemicals such as chlorine, alum, or lime.

There are several questions which must be answered before any preventive procedure can be recommended for the protection of the public. The more important of these are: Is there a health problem; is the procedure effective; is it safe; is it economical; it is readily applied and controlled; is the procedure legal; and, does it have the approval of the qualified professional health workers?

Do we have a dental health problem in the United States?

Dental disease, the most common disease of mankind, begins soon after teeth first appear in the child's mouth and continues to be a major problem throughout life. The importance of dental health in maintaining physical and emotional well-being is recognized by all.

Dental defects are a major school health problem and lead the list of uncorrected defects. Examinations of school children last year in one large city disclosed that 75 percent had dental defects. The next highest listing of defects affected less than 15 percent of the children and included underweight, poor vision, poor posture, overweight, and infected tonsils. A recent survey in a western state disclosed that over one-third of the six-year-olds, three-fourths of the seven-year-olds, and practically all of the nine-year-olds had been attacked by dental decay. Considerably less than half the total needs for dental care in our children

*Presented at the National Congress of Parents and Teachers, San Francisco, May 22, 1956. Reprinted from *California's Health*, September 15, 1956.

are being met. These untreated needs of childhood carry over into later life so that by age 40 about one-fourth of the people have lost all of their teeth.

For a good many years the growth of the dental profession has not kept pace with the population increase in this country. It seems quite unlikely that, in the foreseeable future, there will be enough dentists to care for the dental needs of our population. The only practical solution of this problem lies in the prevention of a major portion of dental disease.

The first question has been answered affirmatively—there very definitely is a health problem.

Is water fluoridation effective in reducing the problem?

Water-borne fluorides are not something new in the United States. Over 65 million people for years have used water containing fluoride naturally—4,500,000 of these drink naturally fluoridated water containing 0.7 or more parts of fluoride per million parts of water. Although one part fluoride to each million parts of water (ppm) is the recommended concentration, many thousands of people for years have used water containing three times, five times, and even seven or more times this amount. Examples of these communities include Thomaston, Alabama, which uses water containing 3.2 ppm; Bureau, Illinois, 5.5 ppm; Langford, South Dakota, 8.0 ppm; Gabbs, Nevada, 8.7 ppm; Gillette, Wyoming, 4.0 ppm; and Bartlett, Texas, 8.0 ppm.

It is interesting to note that as early as 1892 Sir James Critchon-Brown suggested that lack of fluoride in the diet probably was responsible for the high rate of dental decay in England. About 10 years later Dr. J. M. Eager, a Public Health Service physician in Italy, described a condition of the teeth which we now know as dental fluorosis. In the United States studies started in 1908 of a condition known as "Colorado brown stain" laid the foundation for the discovery of the decay preventive value of fluorides in drinking water.

In 1916 two scientists reported "a curious absence of decay" among residents in areas where we now know fluorides were present in the drinking water. However, not until 1931 was it proven that the effective element in the drinking water was the fluoride ion. The presence of 8,500 articles in the scientific literature today attests the vast body of knowledge collected about the fluorides. By 1940 it was proven that insofar as the fluorides are concerned there are about three types of drinking water—those deficient in fluorides, waters with the optimum concentration, and those with excessive amounts of fluoride. Dental decay is extensive in communities using low-fluoride or fluoride-free water while people using drinking water containing the correct amount of fluoride have about two-thirds less tooth decay. The use of water with excessive fluorides results in discoloring or mottling of the teeth. These ranges of fluoride concentration are well within the control of the water plant operator.

A study of adults in two Colorado cities proves that the beneficial effects of fluoride water, accrued during childhood, continue into the older age groups. Boulder, Colorado, uses a fluoride-free water supply while the water supply of Colorado Springs contains 2.5 parts per million of fluoride. In Boulder, 40- to 44-year-old persons had 22 teeth which had decayed, while the same age group in Colorado Springs averaged less than half (10) as many teeth attacked. On comparing the tooth decay rates in these two cities from age 20 to age 45, we note that the decay pattern established in childhood has continued into these older ages. It was found also that the 40- to 44-year group in the fluoride-free city averaged five times as many missing teeth as their counterparts who had used fluoride water all their lives.

In 1945-1946 several studies, each independent of the others, were begun to determine the effectiveness of and the problems involved in adjusting the fluoride concentration of a community water supply which was deficient in this element. Some of these study cities were Grand Rapids, Michigan; Newburgh, New York; Brantford, Ontario; Evanston, Illinois; Marshall, Texas; Sheboygan, Wisconsin; and Lewiston, Idaho. (Let me point out again that each of these studies was conducted independently.)

In Grand Rapids the study was a cooperative project involving the Public Health Service, the State of Michigan,

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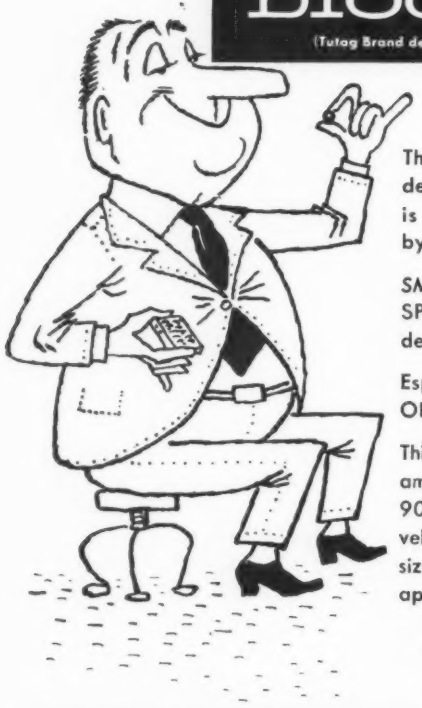

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the University of Michigan, and the City of Grand Rapids. The city has continued to bring the same fluoride-free lake water to the water plant, where since 1945 the fluoride concentration has been adjusted and maintained at one part fluoride per million parts of water throughout the distribution system.

The study has shown that children born in Grand Rapids since 1945 and raised on the fluoride water have received the greatest benefits—about 65 percent less tooth decay than children of the same age prior to fluoridation. The children born before the water was fluoridated obtain less, though significant, reductions in tooth decay. For example, children who were seven years old when water fluoridation was instituted and used this water until they were 16 years of age have nearly 30 percent less decay than 16-year-olds who never used fluoride water. This same pattern of reduction in dental decay in the several age groups was present in Grand Rapids, Michigan, after 10 years of water fluoridation. These observations have been substantiated by similar findings in other independent studies, such as Newburgh, New York, and Brantford, Ontario. Data from these study cities indicate that continuous users of water to which the optimum amount of fluoride has been added have the same pattern of lowered tooth decay rates found in those using natural fluoride water under similar conditions.

The foregoing evidence provides an affirmative answer to our second question. Fluoridation certainly is effective—about 65 percent less tooth decay.

Is water fluoridation safe?

The use, for many years, of natural fluoride waters by a large part of our population afforded excellent opportunities to study the effects of such water on human health. One of the first discoveries in this natural laboratory was that developing teeth are very sensitive to excessive water-borne fluorides. People born and raised where the fluoride concentration exceeds 1.5 parts per million in their drinking water develop varying degrees of mottling of the tooth enamel. The milder degrees of mottled enamel are noticeable only to trained observers, but as the excessive fluoride concentration in the water increases the symptoms become more obvious. As mentioned earlier, some communities in this country use drinking water containing five or six or more parts per million of fluoride. At these higher concentrations there usually is serious disfigurement and damage to the enamel. No other clinically significant physical effects have ever been found even at these high concentrations.

The Public Health Service conducted a study on people born and raised in Cameron and Bartlett, Texas. The Cameron water supply contains naturally 0.4 parts of fluoride per million while in Bartlett the water supply contains 20 times that amount, or 8.0 parts per million. Reports were made on laboratory, X-ray, medical, and oral examinations conducted in 1943 and in 1953. Those examined in Bartlett average over 36 years' use of the high fluoride water. A variety of characteristics in the two groups were compared—including arthritic changes, cardiovascular conditions, hearing loss, bone changes, eye conditions, occurrence of tumors or cysts, fractures, blood pressure variations, thyroid abnormalities, kidney stones, and gall stones. Laboratory findings in the blood and urine were also studied and compared. It was found that no clinically significant physiological or functional effects resulted from the prolonged ingestion of water containing excessive fluoride except for mottled enamel.

Studies on bone fracture experience, bone growth, height and weight, skeletal tolerance, excretion and storage of fluorides, and on the general health of people using fluoride waters have shown no health hazards attributable to fluorides. A comparison of mortality rates in fluoride and non-fluoride communities in Illinois produced neither significant differences in the general death rates nor significant differences in the risk of death from specific diseases such as heart disease, cancer, nephritis, and diabetes. Extensive laboratory research with experimental animals has shown no harmful effect at the 1.0 ppm level recommended for fluoridation. Years of use by a variety of industries indicates that fluoridated water does not interfere with any industrial process.

Another study by the Public Health Service compared mortality experience

of 32 pairs of fluoride and nonfluoride cities of 10,000 or more population in 16 states. Fluoride concentrations in the water supplies of the fluoride cities ranged from 0.7 to 4.0 parts per million. The nonfluoride cities used water containing 0.25 parts per million or less. Total populations of over 800,000 in the fluoride cities and over 1¼ million persons in the nonfluoride cities were involved. There was no statistically significant difference between the mortality rates of fluoride and nonfluoride cities for all causes of death or for deaths due to heart disease, cancer, nephritis, intracranial lesions, or cirrhosis of the liver.

To this day not one piece of scientific evidence has ever been presented to prove that water fluoridated at the recommended level is harmful to health. In answer to our third question it can be truthfully stated—fluoridation is safe.

Is adding fluoride to a community water supply an economical method of reducing tooth decay?

The cost of water fluoridation will vary among communities according to local differences in water supplies, industrial use, types of equipment and the chemical used. Compared with the cost of other compounds used in water treatment the cost of fluoridation is very nominal. In one large city the cost of controlled fluoridation using sodium silicofluoride is $1.36 per million gallons of water. Another city using hydrofluorsilicic acid averages $1.03 per million gallons of water. By comparison in these two cities, the average cost per million gallons for alum was $8.49 and $4.73; for chlorine, $2.42 and $1.99; and for lime, $1.64 and $0.30, respectively.

Of perhaps greater interest is the cost to the individual each year or for a lifetime. In the 1,294 communities now using fluoridated water the cost averages about 10 cents per person per year. The 26 million people in these towns are obtaining the benefits of this preventive for a total of $7 each in an average lifetime of 70 years. For about the cost of filling a badly decayed tooth or for much less than the cost of replacing one lost tooth they are preventing the occurrence of a very sizable amount of tooth decay. The additional benefits derived through improved appearance, better health, fewer lost teeth, and improved dental function are equally important.

Surely there can be no doubt regarding the answer to our question on economy. Water fluoridation is very economical.

Can this decay preventive be readily applied and controlled?

The water engineering practices in controlled water fluoridation while specific and accurate are not particularly difficult. The various state health departments have established regulations governing the application of this procedure in local water plants. Usually the state authority reviews the installation plans and issues a permit to the water company. The regulations call for periodic testing at the water plants, samples sent regularly to the state laboratory for check-testing and weekly or monthly reports on the amount of fluoride compound used and the volume of water pumped. The color reaction methods used in determining fluoride concentration vary only slightly from those used for other water testing. Colorimeters, either visual or photoelectric, are available for these tests and are well within the required accuracy. As mentioned earlier the equipment used in applying the fluoride compound to the water is not new to the water plant operator. Similar equipment has been used for years in water treatment. It also might be pointed out that identical equipment is used in other industries where a high degree of accuracy is required. An example is the enrichment of flour with vitamin supplements. No significant problems in adding fluorides or maintaining the desired level have been reported.

Without hesitation we can answer affirmatively that this preventive procedure can be readily applied and controlled at the desired level.

Is the application of this public health measure legal?

As it has with other public health measures the legality of water fluoridation has been questioned over the last several years. The legality of water fluoridation has been tested in the courts of 13 states and in seven of these states, California, Louisiana, North Dakota, Ohio, Oklahoma, Washington, and Wisconsin the cases were carried to the State Supreme Courts. In the California, Lou-

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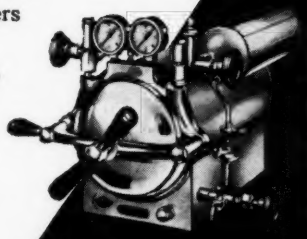

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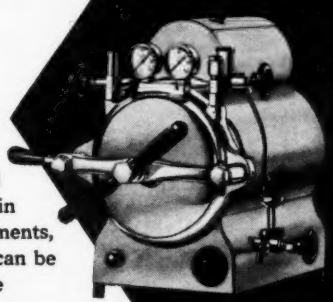

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†"The Management of Viral Hepatitis," by Hyman J. Zimmerman, M. D., Journal of American Academy of General Practice, June 1955

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1. Grayzel, H. G., and Schapiro, S.: Western J. Surgery, Obstet. & Gyn., Oct. 1956.

isiana, and Oklahoma cases appeals were taken to the United States Supreme Court which refused to review them. Fluoridation has been challenged most frequently as an unconstitutional invasion of individual liberties.

In no instance has fluoridation lost a final court action. It is a proper subject for community action. It is legal.

Does water fluoridation have the approval of the qualified health professions?

During the past several years the majority of national health organizations have endorsed water fluoridation as a safe and effective method for reducing the incidence of tooth decay. These national associations, representing the majority of dentists, physicians, and public health scientists in this country, did not give their approval lightly. The endorsements were given only after careful, detailed study of the evidence by special committees, competent members, and expert consultants. A partial list of these national health groups includes, the American Dental Association, the American Medical Association, the National Research Council, the Department of Defense, the United States Public Health Service, the American Public Health Association, the American Academy of Pediatrics, the American Hospital Association, and the Association of State and Territorial Health Officers. Practically all state and local medical and dental societies have given their stamp of approval.

In addition many national lay groups interested in the health and welfare of our citizens have endorsed this great public health procedure and a large majority of their state organizations have followed suit. In 1952 the National Congress of Parents and Teachers approved this health measure.

Last October the New Jersey Congress of Parents and Teachers passed a resolution which emphasized the safety of fluoridation, recognized it as a community measure, and stated:

> Fluoridation of community water supplies shall no longer be regarded by the New Jersey Congress of Parents and Teachers as simply a matter for approval, but that the Congress shall also accept the responsibility at the local level to secure its benefits for our children by initiating such community efforts as suggested by the National Congress of Parents and Teachers, or by cooperating with other local health and welfare organizations and agencies in their efforts to secure fluoridation of community waters as set forth under the regulations of the New Jersey State Department of Health.

Certainly few public health measures have had the solid support that water fluoridation has had from the professional health workers' organizations and from other groups interested in health.

There are, of course, those who oppose fluoridation just as some people even now oppose water chlorination, milk pasteurization, and small pox vaccination. The introduction of water chlorination 40 years ago met with the same type of opposition and essentially the same arguments that are used today against fluoridation. The opposition makes all sorts of claims—but have been unable to produce any evidence that fluoridation is in any way harmful.

The seven questions posed earlier all have been answered in favor of water fluoridation: (1) the dental health problem in this country is so tremendous that prevention is the only solution; (2) water fluoridation is effective—a 65 percent reduction in tooth decay; (3) water fluoridation is safe—extensive laboratory, animal, and human studies prove there is no hazard to health at the recommended level; (4) water fluoridation is economical—for an average of 10 cents per person per year a community can benefit from this public health measure; (5) it is readily applied and controlled—no problems of operation or maintenance of the desired fluoride level have been reported; (6) the method is legal—the courts of 13 states where fluoridation was contested have upheld it as a valid community activity involving no infringement of personal liberty; (7) it has the overwhelming support of qualified professional health workers and other groups interested in the health and welfare of our people.

Fluoridation of drinking water has emerged as one of the outstanding public health developments of recent years. It will take its place in history with other public health achievements.

Health and appearance are of primary concern in our present day culture. Those with lowered resistance or unsightly decayed or missing teeth are handicapped in attaining their place in our society.

The cost of caring for their dental needs is a serious drain on the purses of many.

Certainly a preventive procedure which has successfully passed the test of our seven questions, which offers improved health and appearance and which lowers the need for dental care deserves a high priority in any forward-looking community.

Education for parenthood*

Modern education for parenthood centers around the family rather than the mother and her infant. Today's classes for families expecting another child now include not only the father but the children, offer shared learning for the greatest sharing experiences in life, seek to expand the boundaries of family feeling, and foster an environment conducive to psychic growth for parents and children.

Apparently, the enthusiastic response to our classes at the Maternity Center in New York City and in other large centers of population coincides with a widespread desire for help. Our classes consistently attract more applicants than there are chairs available. Sometimes, young couples apply even before a child is expected.

In part, we attribute the favorable response to the fact that young couples wish to be equal to parenthood. They feel a need to develop their inner resources. They wish to learn what changes to expect in their relationship to each other and in their pattern of living.

Few youthful parents know what they should about human reproduction. Although the majority in our classes have attended college, they have little real knowledge of how a baby is born. Confronted with the process of birth, they wish to understand rather than wonder. Those able to assimilate medical knowledge seek the help of experts. But rather than imprecise answers, they seek facts from which to make their own decisions. They are not willing to accept a passive role. This attitude is perhaps more crystallized in maternity care than in other medical services.

Only 25 years ago, emotional security was a concept limited to psychiatrists and other professional workers. The parents of today, conscious of their own personality disturbances, try to create a good emotional climate for their children. They are irrevocably committed to the momentous adventure of parenthood, and they are unsure of their ability to live it well. They need not only to learn the elements of bathing and diapering an infant. They need help also in understanding their own and the baby's behavior. They wish to avoid the mistakes they feel their parents made unwittingly.

Unlike their parents, who often felt they had to possess a house and car before they could afford babies, today's young people base their security on the family and its social value rather than on material things. Whatever adds social value to the family strengthens their sense of security. Since formal education is an important symbol of ego value in our culture, the undervalued role of parent gains in prestige when it is approached by the educational route.

INDIVIDUAL GOALS AND ASPIRATIONS

Education for parenthood should help the mature individual to derive the utmost satisfaction from the experience and to share that satisfaction with others in the family. It should help the personality to mature in proportion to the complex responsibilities of parenthood. For the unborn child, it should prepare a suitable home.

Education for parents should emphasize the primacy of the home since the disruption of family life is at the root of much unhappiness. It should encourage a broad conception of the family unit to provide the child with opportunities for effective relationships and for learning to live with others. It should provide learning of permanent value so that parents continue to build for happy, healthful living long after they have forgotten

*Brief of paper presented at 16th Eastern States Educational Conference, New York City, April 1956, by Hazel Corbin, R.N., general director, Maternity Center Association, New York City. Reprinted from *Public Health Reports*, October 1956.

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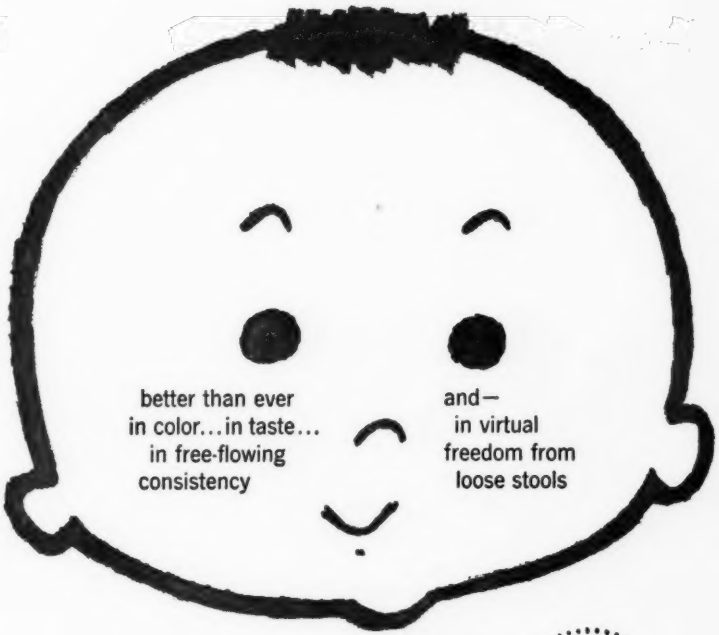

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the details learned in classes for parenthood.

To be effective, health education must be acceptable to people. It is important therefore to help the individuals in the group work out their own methods of achieving what they desire. Although a core of pertinent information should be given, it is not desirable to insist on a particular pattern of performance or care. Effective education keeps in mind that individuals and groups are unique in goals, aspirations, and working methods.

By keeping the class small and having it meet over a fairly long period of time, it is possible to practice permissiveness, both in the teaching pattern and in the conduct of discussion. Young people are accustomed to frank talk, with each other and their friends. They are most receptive when the classroom discussion is at this level of freedom. As they become acquainted, the discussion grows in freedom. This spontaneous talk provides the teacher with the key to unexpressed and unformulated feelings.

Today's parents want to know about fertility and infertility, the uses and dangers of anesthesia and analgesia, the pros and cons of natural childbirth and rooming-in, and the psychophysical rationale of breast feeding. Many want movies of an actual birth.

A good educational program gives them the best available information on which to base their choices. The nature of the child, even before birth, is emphasized throughout so that parents are prepared to receive a baby as an individual personality and not merely as their creature, however loved, to be reared in the pattern of their personalities and wishes.

Often parents are far ahead of professional workers in their ideas of what they should learn. In controversial areas, it is wise to explain the difficulties that may prevent full realization of their desires so that they may adjust sensibly when their efforts fall short of complete success.

Whether they like it or not, for example, most women are obliged to have their babies in hospitals. Whether they like it or not, they are usually separated from their husbands during labor and from their babies after birth. If they make a choice between hospital and home based on realistic information, they are usually able to try sensibly to gain the advantages of both and to adjust without trauma if they don't.

SHARING OF ATTITUDES

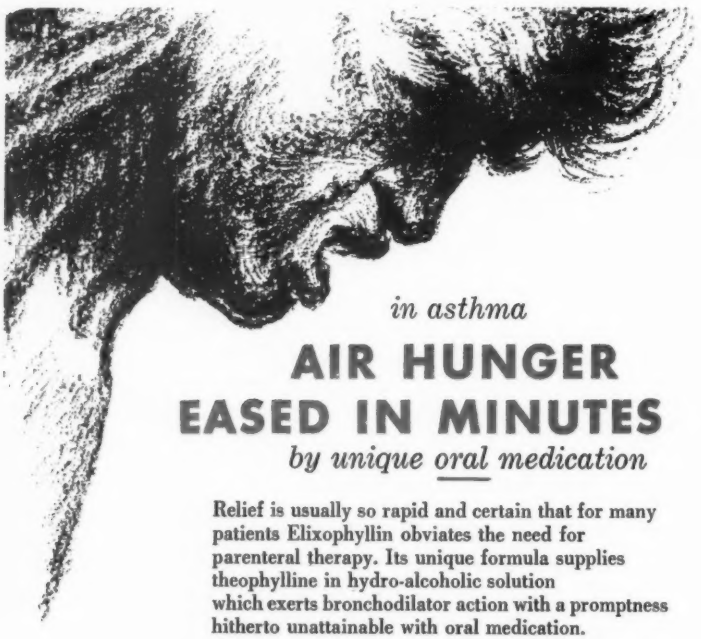
Naturally, all of the initiative does not rest with the class. A good discussion leader creates interest in what should be learned and brings about coincidence of teaching and learning goals. In an early session on intrauterine development, for example, we explain the baby's dependence on the mother and her food intake for the baby's body-building needs. Then parents are ready to receive the session on nutrition with real interest in the child's welfare as well as their own.

In teaching nutrition, we explain how different food elements serve the body. We do not say "eat this or that" but help each mother and father achieve good nutrition within the framework of familiar food habits and tastes.

Even though deliberate attempts at psychotherapy should be avoided, there can be no doubt that the airing of hopes and fears is beneficial. Throughout pregnancy, for example, there is a shift of interest back and forth between the child and self. Expectant parents often feel guilty and abnormal when their beginning parental attitudes give way to self-concern. They do not always realize that parental feeling is a developing and not a full-born thing. As they understand that a measure of emotional conflict is universal, they grow in self-confidence, and the motivation for learning is strengthened.

No large-scale, continued evaluation of parents' classes has been made, though empirically it seems certain that the shared experience has psychotherapeutic value. Mothers comment on the supportive value of what they learned in class. They write of the pleased surprise and appreciation of their physicians at their performance during labor. Nurses report that women who have had prenatal preparation have an easier delivery and react better psychologically. Mothers and mothers-in-law remark on the emotional growth observed in their sons and daughters.

Education for childbearing does not pretend to eradicate neurotic attitudes or deep-rooted personality traits. It does seek to minimize or prevent new traumatic experiences, to help develop insights conducive to a favorable environ-



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ment for the coming child, and to aid in the reconstruction of the family in a society that in many of its values and practices tends to rupture family bonds. Education for parenthood which is focused on the family and its individual and collective needs, and which fosters a secure, happy family setting, contributes to the welfare of society as well as of individual mothers, fathers, and children.

Jones criteria (modified) for guidance in the diagnosis of rheumatic fever*

This report of the Committee on Standards and Criteria for Programs of Care of the Council on Rheumatic Fever and Congenital Heart Disease of the American Heart Association has been approved by the executive committee of that council. The original report was published in "Modern Concepts of Cardiovascular Disease," vol. 24, September 1955. In revised form, it is being given wide circulation to support efforts to detect and treat rheumatic fever.

Rheumatic fever is related to previous infection with group A beta hemolytic streptococcus, but the mechanism of the disease is unknown. Its boundaries are indefinite, and its differentiation from other diseases is sometimes impossible. There is no specific laboratory diagnostic test. The diagnosis must therefore be arbitrary and empirical. Criteria herein set forth are aimed at identifying those individuals who have had or are having an attack of rheumatic fever. They make no attempt to measure rheumatic activity at any given time or to diagnose inactive rheumatic heart disease. Thus, following the designation of an illness as rheumatic fever, the existence of continued activity or the presence of inactive rheumatic heart disease may be indicated by criteria different from those outlined below.

Criteria are necessary in order to minimize both overdiagnosis and underdiagnosis. The tendency to label as rheumatic fever a chronic febrile illness for which no obvious cause can be found is to be deplored. The tragedy which may lie in the wake of the false diagnosis of rheumatic fever may be even greater than the possible harm of missed recognition in questionable cases. The institution of effective prophylactic regimens requiring prolonged administration of sulfadiazine or antibiotic agents places a grave responsibility on the physician in the diagnosis of this illness.

In this statement, the diagnostic features of the disease are divided as originally proposed by Jones into major and minor categories dependent upon their relative occurrence in rheumatic fever and in other disease syndromes from which this disease must be differentiated. Thus chorea is included among the major criteria while fever, a symptom common to many diseases, is placed in a minor category. These major and minor categories have no significance beyond their diagnostic import either as to prognosis, amount of "rheumatic activity," or severity of acute illness. Indeed, a severe manifestation of rheumatic fever such as rheumatic pneumonia is not included because it is difficult to differentiate from congestive cardiac failure and because it almost always occurs in patients whose rheumatic fever is so obvious as to offer no difficulty in diagnosis.

The presence of two major criteria or one major and two minor criteria indicates a high probability of the presence of rheumatic fever with one notable exception (see "other manifestations"). In addition to the major and minor criteria to be used in the recommended formula, other manifestations have been listed which may be used to support the diagnosis. These criteria are not meant to substitute for the wisdom and judgment of the clinician. They are designed only to guide him toward a diagnosis of the disease with the suggestion that he follow carefully all questionable cases and restrict the diagnosis of rheumatic fever to illnesses which meet acceptable criteria.

MAJOR DIAGNOSTIC CRITERIA

Carditis

Murmurs: The presence of a significant apical systolic murmur, apical middiastolic murmur, or basal diastolic murmur in an individual without a history of previous rheumatic fever or in whom there is good reason to believe there

*Reprinted from *Public Health Reports*, July, 1956.

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1 J.A.M.A. 140:672 (June 25) 1949

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was no pre-existing rheumatic heart disease; or a change in the character of any of these murmurs under observation in an individual with a history of rheumatic fever or rheumatic heart disease.

Increasing Cardiac Enlargement: Obviously increasing cardiac enlargement by X-ray.

Pericarditis: Pericarditis manifested by a friction rub, pericardial effusion, or definite electrocardiographic evidence.

Congestive Failure: Congestive heart failure (in a child or young adult under 25) in the absence of other causes

Polyarthritis

Polyarthritis tends to be migratory and is manifested by pain and limitation of active motion, or by tenderness, heat, redness or swelling of two or more joints. Arthralgia alone without objective evidence of joint involvement is not a major manifestation.

Chorea

This must be differentiated from habit spasm, athetosis, and cerebellar ataxia. Movements must be characteristic, involuntary and of moderate severity if chorea is to be used as a major manifestation.

Subcutaneous Nodules

These are shot-like, hard bodies seen or felt over the extensor surface of certain joints, particularly elbows, knees and wrists, in the occipital region, or over the spinous processes of the thoracic and lumbar vertebrae.

Erythema Marginatum

This recurrent, pink, characteristic rash of rheumatic fever, in which the color gradually fades away from its sharp scalloped edge, is found mainly over the trunk, sometimes on the extremities, but not on the face. It is transient, is brought out by heat and migrates from place to place.

MINOR DIAGNOSTIC CRITERIA

Fever

A significant rise in temperature is a common symptom, but, because it occurs in so many illnesses, it has little differential diagnostic value. In order to be included, the elevation in temperature must clearly exceed the normal diurnal fluctuation in which there is great individual variation.

Arthralgia

Pain clearly located without objective findings is only a minor criterion for diagnosis. The pain must be in the joint, not in the muscles or other periarticular tissues, and must be distinguished from the nocturnal pain in the extremities occurring in normal children. Arthralgia must not be used as a minor criterion when polyarthritis is included as a major criterion.

Prolonged P-R Interval in the Electrocardiogram

Prolongation of the P-R interval may be nonspecific; it is considered a minor criterion and is not diagnostic of carditis. It cannot be used if carditis is already included as a major manifestation.

Increased Erythrocyte Sedimentation Rate, Presence of C-Reactive Protein, or Leukocytosis

Elevation in one or more of these nonspecific tests may be considered as a single minor criterion. Particularly to be deplored is the tendency to use any of these tests as a major criterion or as diagnostic of rheumatic fever. There are many other nonspecific tests, but these three are most commonly used.

Evidence of Preceding Beta Hemolytic Streptococcal Infection

This must be documented by (1) a history of scarlet fever or by a typical clinical picture of other streptococcal infection preceding the onset of rheumatic fever by one week to one month, the nature of the infection being confirmed by a history of immediate contact with other individuals having typical streptococcal infection or by positive culture of the nose or throat in which beta hemolytic streptococcus predominates; or (2) an elevated or rising antistreptolysin-O titer.

Previous History of Rheumatic Fever or the Presence of Inactive Rheumatic Heart Disease

The existence of either of these may be used as a minor criterion to aid in deciding the rheumatic nature of the illness in question. For this use, the previous history must be documented by the same objective criteria as are set forth in this statement or by the presence of inactive rheumatic heart disease.

OTHER MANIFESTATIONS

These include systemic manifestations such as loss of weight, easy fatigability,

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elevated sleeping pulse rate (tachycardia out of proportion to fever), malaise, sweating, pallor or anemia, and local manifestations such as epistaxis, erythema nodosum, precordial pain, abdominal pain, headache, and vomiting. These, as well as a family history of rheumatic fever, provide additional evidence of the presence of rheumatic fever but are not to be included as diagnostic criteria.

There are combinations of these diagnostic criteria which occur in the presence of other illnesses which must be ruled out before a definite diagnosis is made. One combination in particular—polyarthritis, fever, and elevated sedimentation rate—is the weakest of all combinations of major and minor criteria. Diseases to be ruled out include rheumatoid arthritis, gonococcal arthritis, lupus erythematosus disseminatus, subacute bacterial endocarditis, non-specific pericarditis with effusion, leukemia, sickle cell anemia, serum sickness (including manifestations of penicillin sensitivity), tuberculosis, poliomyelitis, undulant fever, and septicemias, particularly meningococcemia.

MURMURS INDICATING CARDITIS

Significant Apical Systolic Murmur

A significant apical systolic murmur is long, filling most of systole; is heard best at the apex; is as well transmitted toward the axilla as over the precordium; and does not change with position or respiration. It must be differentiated from an innocent (functional) murmur which is frequently found in normal people. This innocent murmur is systolic, occasionally harsh, is heard best along the left sternal border and usually changes with position and respiration. Borderline systolic murmurs, intermediate in location and nature, occur and should be carefully watched. Questionable murmurs which are intermittently present or which, after a period of observation, cannot be clearly classified as significant are rarely of any import.

Apical Mid-Diastolic Murmur

A significant organic apical systolic murmur is frequently accompanied by a low-pitched, short mid-diastolic murmur which is sharply localized to the chest wall over the apex of the heart and often heard best with a patient in the left lateral position with the breath held in expiration. This murmur, rarely present in the absence of an apical systolic murmur, confirms the significant nature of the latter. It must be differentiated from the long, low-pitched, crescendo apical presystolic murmur followed by an accentuated mitral first sound which is indicative of mitral stenosis but not of acute carditis.

Basal Diastolic Murmur

The development of a basal diastolic murmur of aortic insufficiency is also indicative of carditis. It is an early, short, diminuendo murmur usually heard only or heard best along the left sternal border in deep expiration. It has great diagnostic value, even though it may be difficult to hear and present only intermittently.

Longevity reaches three score and ten*

A historic milestone was reached in 1955 when the expectation of life at birth among American wage earners and their families, as represented by the Industrial policyholders of the Metropolitan Life Insurance Company, rose to a new high of 70.0 years. Although this figure is only slightly higher than that for 1954, it marks the 12th successive year to record a rise. The average length of life of policyholders has increased 5 years since 1945, 10 years since 1935, and 24 years since 1909. In 1879-94, the earliest period for which data are available in this experience, the average was only 34 years—less than half that for 1955.

Each of the color-sex groups among the insured has shared in the gains of recent years, but not in equal measure. Females have done better than males, with the result that their advantage is greater than ever before. In 1955, the expectation of life at age 5 was 70.1 years for white females and 63.9 years for white males, a difference of more than 6 years. The margin in favor of females decreases with advance in age, but it still amounts to 2½ years at age 65, when the expectation of life is 15.1 years for white females and 12.5 years for white males.

*Reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, September 1956.

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Although the colored policyholders have been making greater gains than the white in recent years, their longevity record still lags behind that of the white policyholders. In 1955, the expectation of life at age 5 for colored males was nearly two years less than that for white males; for females the disparity was as much as four and a half years.

The achievements in life conservation are reflected in the very low death rates now experienced in childhood and early adult life. Mortality rates under 1 per 1,000 were recorded from ages 3 through 16 years among white males and in the age range 2-33 years among white females. The experience among the colored policyholders was not as favorable; death rates under 1 per 1,000 were recorded at ages 4-15 for males and 3-20 for females. The disadvantage of the colored was particularly evident in early and middle adult life. For men at ages 22-35, the mortality among the colored was more than 1½ times that for the white. For women the difference was even greater; from age 23 to the end of the childbearing period the ratio of mortality was at least 2 to 1.

For nearly two generations the average length of life has been increasing more rapidly among American wage earners and their families than for the general population of the United States. In 1909, the average lifetime of the Company's Industrial policyholders was about six years less than that for the population as a whole. Since then the disparity has been narrowing; at present the two groups are on a par.

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Books received

Books received for review during the period from October 5 to November 5, 1956, are listed below. Reviews will be published as space permits.

THE MANAGEMENT OF FRACTURES, DISLOCATIONS AND SPRAINS. By John Albert Key, B.S., M.D., F.A.C.S., St. Louis, Missouri, Clinical Professor Emeritus of Orthopedic Surgery, Washington University School of Medicine; Associate Surgeon, Barnes, Children's and City Hospitals; and H. Earle Conwell, M.D., F.A.C.S., Birmingham, Alabama, Associate Professor of Orthopaedic Surgery, University of Alabama School of Medicine; Associate Orthopaedic Surgeon, University Hospital; Attending Orthopaedic Surgeon, St. Vincent's Hospital, Baptist Hospitals; Chief of the Orthopaedic Service, South Highland Infirmary; Consulting Orthopaedic Surgeon, Veteran's Hospital, Tuscaloosa, Alabama, and Carraway Methodist Hospital, Birmingham, Alabama; Chief of the Conwell Orthopaedic Clinic, Birmingham, Alabama; Member of the Alabama State Crippled Children's Orthopaedic Advisory Board. Ed. 6. Cloth. Pp. 1168, with illustrations. Price $20.00. The C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, 1956.

DISEASES OF THE HEART. By Charles K. Friedberg, M.D., Attending Physician, The Mount Sinai Hospital, New York; Associate Clinical Professor of Medicine, College of Physicians and Surgeons, Columbia University. Ed. 2. Cloth. Pp. 1161, with illustrations. Price $18.00. W. B. Saunders Company, W. Washington Square, Philadelphia 5, 1956.

PRACTICAL PEDIATRIC DERMATOLOGY. By Morris Leider, M.D., Associate Professor of Dermatology and Syphilology, New York University Post-Graduate Medical School; Visiting Physician, Bellevue Hospital; Associate Attending Physician, University Hospital, New York University-Bellevue Medical Center; Diplomate of the American Board of Dermatology and Syphilology. Cloth. Pp. 433, with illustrations. Price $10.50. The C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, 1956.

DISEASES OF THE BREAST. By C. D. Haagensen, M.D., Professor of Clinical Surgery, The College of Physicians and Surgeons, Columbia University; Director of Surgery, The Francis Delafield Hospital, Columbia-Presbyterian Medical Center. Cloth. Pp. 751, with illustrations. Price $16.00. W. B. Saunders Company, W. Washington Square, Philadelphia 5, 1956.

ESSENTIALS OF HISTOLOGY. By Margaret M. Hoskins, Ph.D., and Gerrit Bevelander, Ph.D., New York University. Ed. 3. Cloth. Pp. 254, with illustrations. Price $4.00. The C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, 1956.

CLINICAL EXAMINATIONS IN NEUROLOGY. By Members of the Sections of Neurology and Section of Physiology, Mayo Clinic and Mayo Foundation for Medical Education and Research, Graduate School, University of Minnesota, Rochester, Minnesota. Cloth. Pp. 370, with illustrations. Price $7.50. W. B. Saunders Company, W. Washington Square, Philadelphia 5, 1956.

DIABETES MELLITUS. Handbook for Physicians. By Howard F. Root, M.D., Sc.D., F.A.C.P., Medical Director of the Joslin Clinic, Boston; Lecturer in Medicine, Harvard

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CLINICAL ROENTGENOLOGY. Volume IV. The Digestive Tract, the Gall Bladder, Liver and Pancreas, the Excretory Tract and Special Studies Emphasizing Differential Considerations. By Alfred A. de Lorimier, M.D., Radiologist, Saint Francis Memorial Hospital, San Francisco, California, Consultant in Radiology for the United States Army, at the Letterman Army Hospital; Consultant in Radiation Therapy for the United States Public Health Service at the U.S. Marine Hospital, San Francisco, California; Formerly, Commandant, The Army School of Roentgenology; and Henry G. Moehring, M.D., Radiologist, Duluth Clinic, Duluth, Minnesota; Formerly, Director, The Army School of Roentgenology; and John R. Hannan, M.D., Radiologist, Cleveland, Ohio; Radiologist, Lake County Memorial Hospital, Painesville, Ohio; Formerly, Director, The Army School of Roentgenology; Associate Professor, Diagnostic Roentgenology, The Frank E. Bunts Educational Institute, Cleveland Clinic Foundation; Staff, Department of Roentgenology, Cleveland Clinic Foundation. Cloth. Pp. 676, with illustrations. Price $24.50. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1956.

SICK CHILDREN—Diagnosis and Treatment. By Donald Paterson, M.D. (Edin.), F.R.C.P. (Lond.), F.R.C.P. (Canada), Consulting Physician to The Hospital for Sick Children, Great Ormond Street, London; Consulting Paediatrician, Westminster Hospital, London; Honorary Consultant to the Department of Paediatrics at the Vancouver General Hospital; Sometime Clinical Professor of Paediatrics, Faculty of Medicine, University of British Columbia and Senior in Paediatrics, Vancouver General Hospital. Revised by Reginald Lightwood, M.D. (Lond.), F.R.C.P. (Lond.), D.P.H. (Eng.), Director, Paediatric Unit, St. Mary's Hospital Medical School, University of London, and Physician-in-Charge, Children's Department, St. Mary's Hospital, London; Physician to The Hospital for Sick Children, Great Ormond Street, London; Paediatrician to the Research Unit for Juvenile Rheumatism, Canadian Red Cross Memorial Hospital, Taplow; External Examiner in Paediatrics to Memorial Hospital, Taplow; External Examiner in Paediatrics to the University of Wales. With the assistance of F. S. W. Brimblecombe, M.D. (Lond.), M.R.C.P. (Lond.), D.C.H., Paediatrician, Royal Devon and Exeter Hospital, and Exeter City Hospital; Consultant Paediatrician, Exeter Clinical Area. Ed. 7. Cloth. Pp. 593, with illustrations. Price $8.75. (North American market only) J. B. Lippincott Company, E. Washington Square, Philadelphia 5, 1956.

NEUROLOGY OF THE OCULAR MUSCLES. By David G. Cogan, M.D., Professor of Ophthalmology, Harvard Medical School; Director, Howe Laboratory of Ophthalmology, Boston, Massachusetts. Ed. 2. Cloth. Pp. 296, with illustrations. Price $8.50. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1956.

HANDBOOK OF PEDIATRIC MEDICAL EMERGENCIES. By Adolph G. DeSanctis, M.D., Professor of Pediatrics and Chairman of the Department of Pediatrics, Post-Graduate Medical School, New York University-Bellevue Medical Center; Director of Pediatrics, University Hospital, New York University-Bellevue Medical Center; Director of Pediatrics, Gouverneur Hospital, New York City. With the collaboration of Charles Varga, M.D., Portland, Oregon, and Ten Contributors. Ed. 2. Cloth. Pp. 389, with illustrations. Price $6.25. The C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, 1956.

PHARMACOLOGY AND ORAL THERAPEUTICS. A Textbook for Students and Practitioners. By Edward C. Dobbs, B.S., D.D.S., F.A.C.D., Professor of Pharmacology and Therapeutics, Dental School, University of Maryland. Ed. 11. Cloth. Pp. 579, with illustrations. Price $9.00. The C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, 1956.

POSTURAL AND RELAXATION TRAINING IN PHYSIOTHERAPY AND PHYSICAL EDUCATION. By John H. C. Colson, F.C.S.P., M.S.R.G., M.A.O.T., Principal, School of Remedial Gymnastics and Recreational Therapy, and Director of Rehabilitation, Pinderfields Hospital, Wakefield. Foreword by J. M. P. Clark, M.B.E., M.B., Ch.B., F.R.C.S. Cloth. Pp. 105, with illustrations. Price $2.50. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1956.

CHEMOSURGERY IN CANCER, GANGRENE AND INFECTIONS. Featuring a New Method for the Microscopically Controlled Excision of Cancer. By Frederic E. Mohs, B.Sc., M.D., Associate Professor of Chemosurgery, Department of Surgery, University of Wisconsin Medical School; Head of the Chemosurgery Clinic, State of Wisconsin General Hospital; Research Associate, McArdle Memorial Laboratory for Cancer Research, Madison, Wisconsin. Cloth. Pp. 305, with illustrations. Price $13.50. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1956.

PRACTITIONERS' CONFERENCES Held at The New York Hospital-Cornell Medical Center. Volume 4. Edited by Claude E. Forkner, M.D., F.A.C.P., Professor of Clinical Medicine, Cornell University Medical College; Attending Physician, The New York Hospital; Consultant in Medicine (Hematology), Roosevelt Hospital. Cloth. Pp. 407, with illustrations. Price $6.75. Appleton-Century-Crofts, 35 W. 32nd St., New York 1, 1956.

PELVIMETRY. By Herbert Thoms, M.D., Emeritus Professor of Obstetrics and Gynecology, Yale University School of Medicine, New Haven, Connecticut. Cloth. Pp. 120, with illustrations. Price $5.00. Paul B. Hoeber, Medical Book Department of Harper & Brothers, 49 E. 33rd St., New York 16, 1956.

DYNAMIC PSYCHIATRY IN SIMPLE TERMS. By Robert R. Mezer, M.D., Senior Staff Psychiatrist, Community Clinic, Massachusetts Mental Health Center and Harvard Medical School. Foreword by Harry C. Solomon, M.D., Medical Director, Massachusetts Mental Health Center, and Professor of Psychiatry, Harvard Medical School. Paper. Pp. 174. Price $2.50. Springer Publishing Company, 44 E. 23rd St., New York 10, 1956.

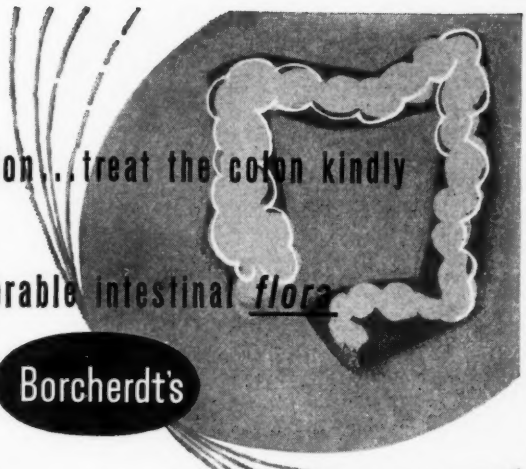
UROLOGY AND INDUSTRY. By Leonard Paul Wershub, M.D., F.A.C.S., F.I.C.S., D.I.C.S., D.U., Associate Professor of Urology, New York Medical College, Metropolitan Medical Center, New York, New York. Cloth. Pp. 151. Price $5.00. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1956.

THEY CALLED HIM DOCTOR. By Marsh Morrison. Author of City of Fingerless Men, etc. Cloth. Pp. 349. Price $4.95. Frederick Fell, 386 Fourth Ave., New York 16, 1956.

GLAMOUR, GLUCOSE AND GLANDS. By Dr. Frank J. Wilson. Cloth. Pp. 163. Price $3.00. Vantage Press, 120 W. 31st St., New York 1, 1956.

SCIENCE AND SURGERY. By Dr. Frank G. Slaughter. Paper. Pp. 272. Price 35c—Perma Book edition. Pocket Books, 630 Fifth Ave., New York 20, 1956.

COMPARATIVE ANATOMY OF THE EYE. By Jack H. Prince, F.B.O.A., F.S.M.C., F.R.M.S., F.Z.S. (London), Assistant Research Professor, Department of Ophthalmology, University Hospital, Ohio State University, Columbus, Ohio. Cloth. Pp. 418, with illustrations. Price $8.50. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1956.



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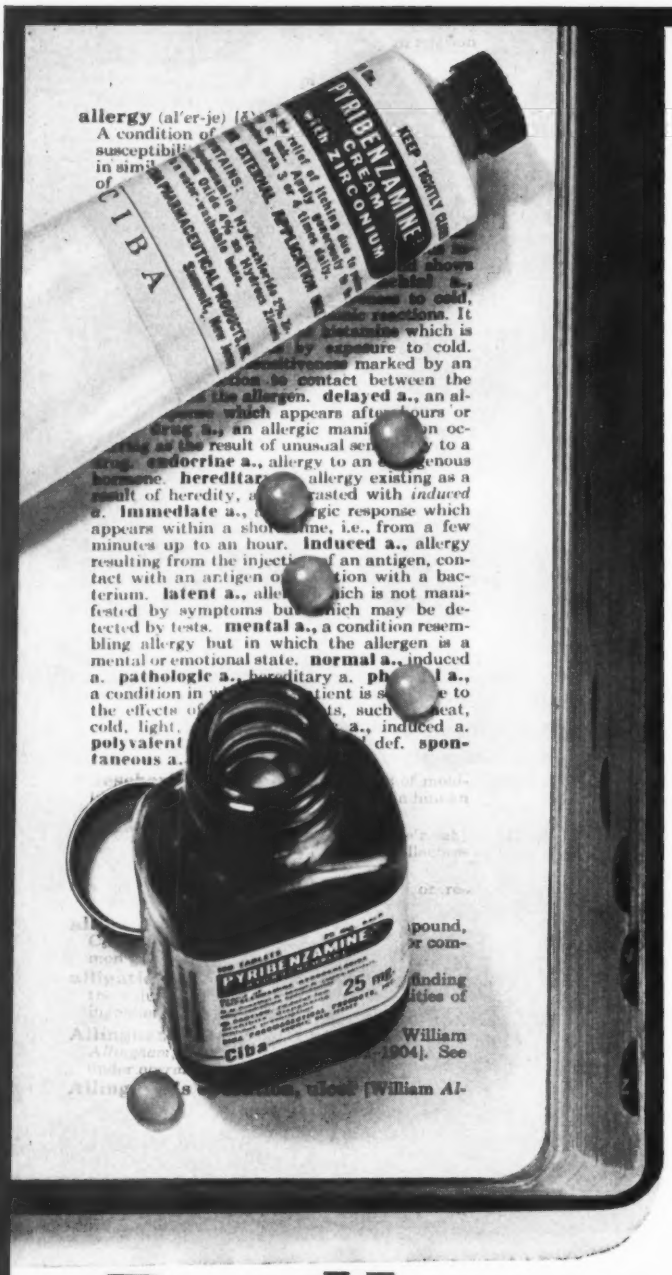

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1. Lovejoy, H. B., Feinberg, S. M., and Canterbury, E. A.: J. Allergy *20*:350 (Sept.) 1949. 2. Goodman, L. S., and Gilman, A.: *The Pharmacological Basis of Therapeutics*, 2nd Ed., The Macmillan Company, New York, 1955, p. 660.

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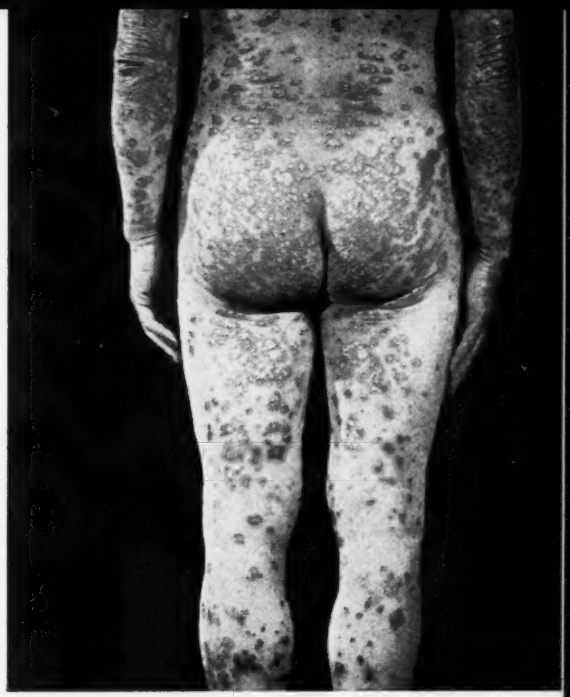

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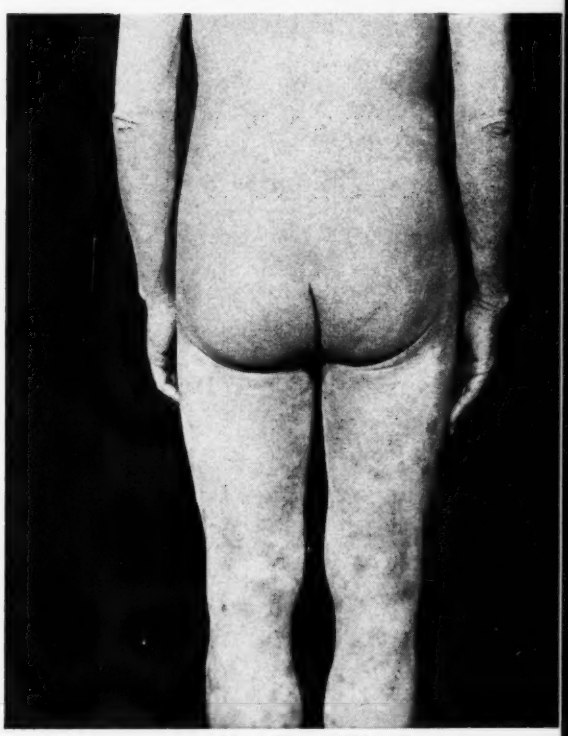

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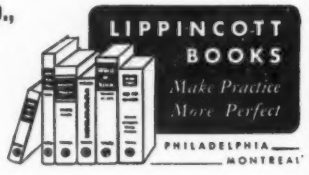

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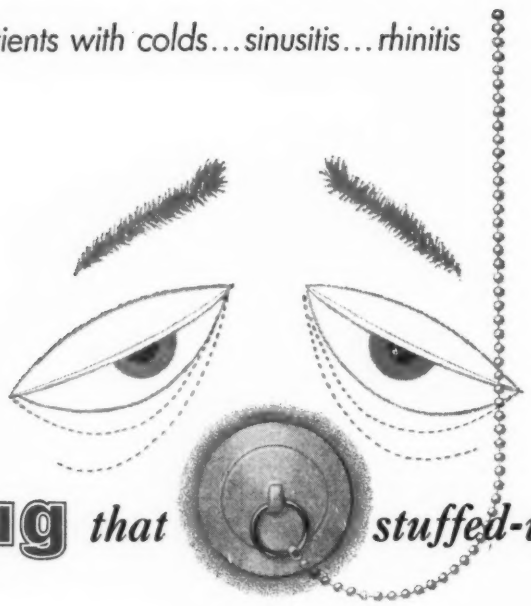

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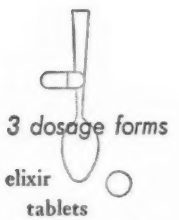

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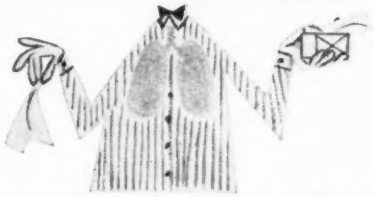

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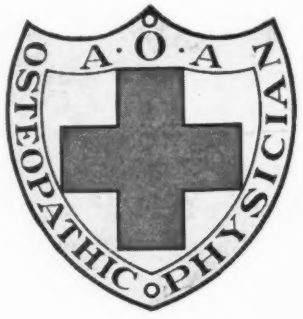

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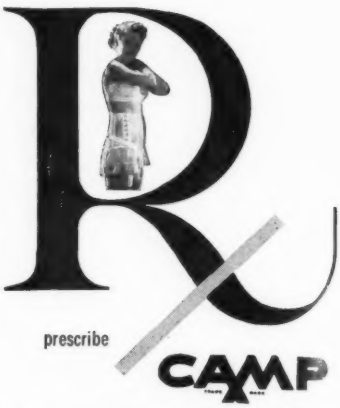

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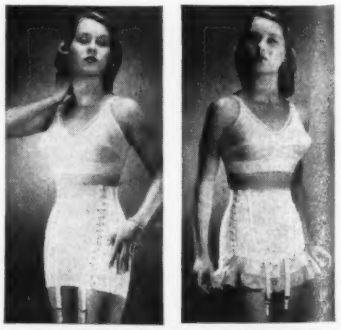

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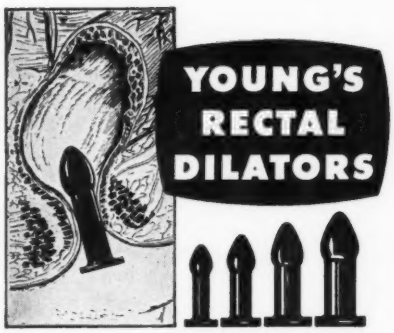

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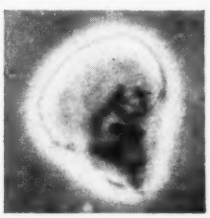

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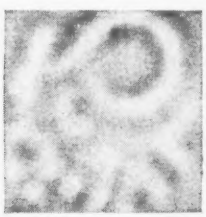

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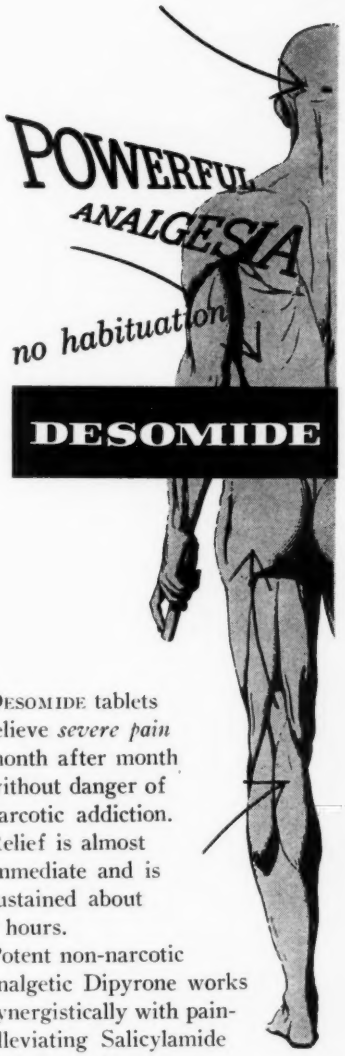


DESOMIDE tablets relieve *severe pain* month after month without danger of narcotic addiction. Relief is almost immediate and is sustained about 7 hours.
Potent non-narcotic analgetic Dipyrone works synergistically with pain-alleviating Salicylamide and mood elevating dl-Desoxyephedrine Hcl. In many cases you can substitute DESOMIDE for morphine, codeine, and other habit-forming narcotics and barbiturates.

Desomide samples and literature on request.

Indications: arthritis, neuritis, musculoskeletal pain, biliary and renal colic, gout, bursitis, inflammation, childbirth, childbirth afterpains, and other painful symptoms.

Desomide Mallard: white round, divided tablet containing Dipyrone 100 mgs., Salicylamide 100 mgs., dl Desoxyephedrine Hcl 1.5 mgs.

AVAILABLE: Bottles, 100, 1000.

There's always a Leader

MALLARD, INC.

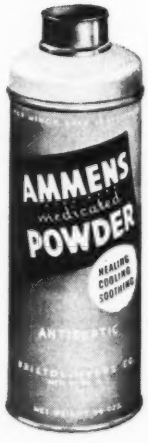
3021 WABASH, DETROIT 16, MICHIGAN

baby's best bodyguard

AMMENS® gives soothing protection to babies' tender skin. And when diaper rash, prickly heat or other skin irritations make him restless, AMMENS eases discomfort, promotes healing.

AMMENS contains starch, widely and uniformly dispersed in talc for maximum absorption of moisture; oxyquinolin and zinc oxide protect macerated crevices against bacterial invasion.

AMMENS is fluffy, white, and smells good, too. Tell baby's mother to keep a can of AMMENS handy.

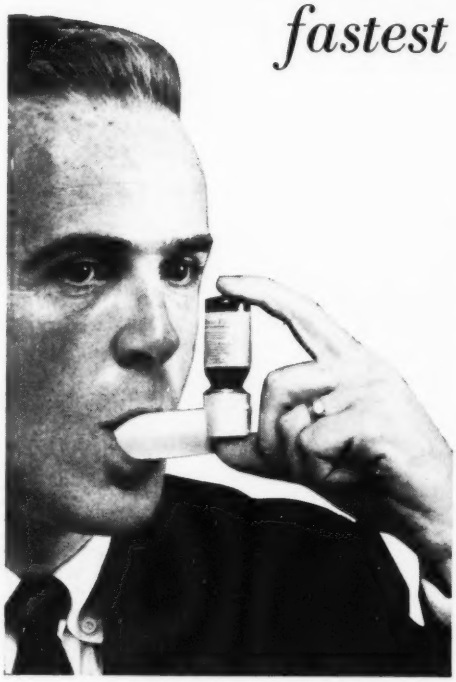


AMMENS® *MEDICATED* POWDER

Bristol-Myers Company, 19 West 50 Street, New York 20, N. Y.
Distributor for Charles Ammen Co., Alexandria, La.

In Angina Pectoris

Medihaler-Nitro



fastest relief of the acute attack

MEDIHALER-NITRO is octyl nitrite (1%) in aerosol solution; delivered by metered-dosage nebulization, using the lungs as portal of entry, it assures fastest relief and prolonged effect; it is free from disagreeable, irritating odor, and less apt to produce side actions than are nitroglycerin and amyl nitrite.

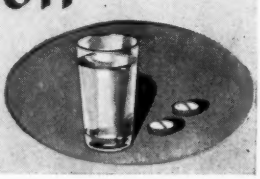
To be used only with the MEDIHALER® ORAL ADAPTER made of unbreakable plastic with no moving parts. Medication and Adapter fit into pocket-size plastic carrying case. One or two inhalations provide prompt relief of an attack of angina pectoris.

MEDIHALER...*The New Measured-Dose Principle of Nebulization*

and for definitive therapy... fewer and fewer attacks of less and less intensity

Long-acting tablets containing pentaerythritol tetranitrate (PETN) 10 mg. and Rauwiloid® (alseroxylon) 1 mg. reduce the incidence and intensity of attacks and lead to objective improvement demonstrable by ECG. Dosage: one or two tablets q.i.d., before meals and on retiring

Pentoxylon®



Riker

LOS ANGELES

when blood pressure must come down

Feb. 1, 1956
Mrs. B.R. age 52 housewife
wt. 159 pulse 85 B.P. 210/116
Complains of "nervousness";
visual disturbances. Last seen
10/27/55 B.P. 180/94
Retina – slight nicking,
ordered c.b.c. and urinalysis
℞ Priming with Serpasil 0.5 mg. daily.
In few days stop Serpasil, start
Serpasil-Apresoline #2 tid q.i.d.

Serpasil®-Apresoline®

hydrochloride

(reserpine and hydralazine hydrochloride CIBA)

When more than the central antihypertensive effect of Serpasil alone is needed to lower blood pressure, you will often see gratifying response to the combined antihypertensive action of Serpasil-Apresoline. And because Apresoline is effective in lower dosage when combined with Serpasil, there is a minimum of side effects.

NOTE: *All patients to be given Serpasil-Apresoline may benefit from priming therapy with Serpasil.*

CIBA
SUMMIT, N. J. 2/22834

SUPPLIED: *Tablets* #2 (standard-strength, scored), each containing 0.2 mg. Serpasil and 50 mg. Apresoline hydrochloride; *Tablets* #1 (half-strength, scored), each containing 0.1 mg. Serpasil and 25 mg. Apresoline hydrochloride.